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MALIGNANT MELANOMAS*

A Clinical Study

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OUR interest in malignant melanomas has been stimulated by the extreme pessimism expressed toward the disease by many members of the medical profession. Malignant melanomas have been recognized as probably the most highly malignant of all tumors, and we are aware of many cases in which a hopeless prognosis has been given, and for this reason no treatment advised. Furthermore, many physicians still believe that intervention of any sort will result in widespread metastases. We have been eager to learn through a clinical study of a series of cases whether surgical measures have been effective. Records totaling 174 have been reviewed. Of these, 74 were from the Pondville Hospital, 88 from the Massachusetts General Hospital, 3 from the Cancer Section of the Westfield Sanatorium, and 9 were those of private cases.

We are using the term "malignant melanoma," as recommended by Masson,[1] to represent the tumors variously called melanotic sarcomas, melanosarcomas or melanocarcinomas. Masson has shown that these pigment cells are ectodermal in origin and arise from cells derived from the neural crest. While the lesions are most frequently associated with the skin, they may arise in any part of the body, as cases of primary lesions occurring in practically all structures have been reported.[2–7] Those in the skin usually contain a blue-black or intense black pigment which enables one to make a clinical diagnosis. There are many variations in the pigmentation, and some cells contain no pigment at all. The few cases of amelanotic malignant melanomas showed no difference in course or results from those with pigment. Lesions may be flat or raised above the skin surface and are rarely pedunculated.

No marked difference was noted in the sex incidence, as there were 79 men and 95 women in our series. Cases were found in patients of extremely diverse ages, the youngest being seven and the oldest eighty-eight; the greatest incidence was in the age group from sixty to sixty-nine (Table 1).

TABLE 1. *Age Incidence.*

AGE	NO. OF CASES
1–9	1
10–19	4
20–29	14
30–39	15
40–49	34
50–59	32
60–69	46
70–79	24
80–89	4
Total	174

Occupation was not a factor, either in the incidence or in the course of the disease.

The distribution of the lesions was of interest (Table 2). Eighty per cent of the tumors occurred

TABLE 2. *Distribution of Lesions.*

DISTRIBUTION	NO. OF CASES	PER CENT
Lower extremity	55	31.6
Upper extremity	11	6.4
Eye	25	14.4
Face and neck	41	23.5
Trunk	28	16.1
Groin and axilla (probable lymph node)	4	2.3
Meninges	3	1.7
Anus	3	1.7
Salivary glands	2	1.1
Vulva	1	0.6
Scalp	1	0.6
Total	174	

on the surface of the body, where they were readily visualized. In nearly a third of our cases they were found on the lower extremities. We wish to stress the high incidence of malignant melanomas on the feet and legs. Those in the Pondville Hospital group represented 55 per cent of all the malignant tumors on the skin of the lower extremities,

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and 35 per cent of all tumors, benign and malignant, in this location.

There were 25 cases where the primary focus was in the uveal tract of the eye. Only 3 of these tumors were seen as primary lesions. In many of the secondary cases at the Massachusetts General Hospital the patients had received primary treatment at the Massachusetts Eye and Ear Infirmary, and came to our clinic only after metastatic disease had appeared. For this reason it is quite possible that we do not have as high a percentage of eye cases in our series as would normally be found in the community.

Frequent sites for this condition were the face, neck and trunk. On the trunk, the majority appeared in the region of the scapula. We have no explanation of this fact, and mention it only to emphasize the importance of considering malignancy in tumors in this area. Adair,[8] in his study of 400 cases, states that as a rule the lesion is congenital, but in a few cases there was no previous lesion until a short time before the appearance of the malignant melanoma. Our series has failed to confirm this statement, for in only 18 per cent of the records in which accurate data were found was there a history of a birthmark. Furthermore, 33 per cent of the patients had had a lesion for less than one year (Table 3). A history of a recent change in a lesion of considerable duration was noted in 54 cases and was considered of diagnostic importance. No instance of malignant change in a hairy mole was noted in this series. We have seen such an occurrence in one patient very recently.

TABLE 3. *Duration of Lesions.*

DURATION	NO. OF CASES	PERCENTAGE OF KNOWN CASES
Present at birth	24	18.8
Less than 1 year	42	32.8
1–3 years	29	22.6
3–5 years	8	6.3
5 years or over	25	19.5
	128	
Unknown	46	
Total	174	

Trauma has frequently been mentioned as an exciting factor in the stimulation of dormant lesions. Our investigation substantiates this in many cases (Table 4). In this disease on the foot, 8 patients gave a definite history of irritation by a nail or a tight shoe; we believe that it may be a factor in all the foot lesions. Nine patients gave a history of having had desiccation or treatment with caustics followed by an immediate proliferation of the growth. Wherever there is a possibility of a mole's being a benign or malignant melanoma, Ewing[9] states that it seems advisable not to use electrodesiccation or a local cauterizing agent. Amadon[10] reports 27 cases treated with electrocoagulation, with 100 per cent recurrence. Biopsy, too, is a dangerous procedure, and total removal should be practiced in a suspicious lesion if one is seeking a pathological diagnosis. In 24 cases rapid growth followed injury by incision, a direct blow or fall. One case illustrates this well. A patient experienced a chip fracture of a cervical vertebra by a fall. Two years later she developed neurological symptoms, and operation revealed a malignant melanoma involving the dura and cord at the site of fracture.

TABLE 4. *Trauma as an Exciting Cause.*

SOURCE OF TRAUMA	NO. OF CASES
Electrodesiccation or use of caustics	9
Irritation by clothing	5
Shoes (definite history in 8 cases)	32
Mechanical (incision, blow, fall, etc.)	24
None	104
Total	174

We have found that it is not necessary to have a pre-existing benign lesion or birthmark for the origin of a malignant melanoma. Many of these tumors in our series were apparently malignant from the onset. Inasmuch as pigmented moles are exceedingly common and many must be subjected to irritation, it follows that a comparatively small percentage of them ever develop into malignant tumors. When changes do occur in a mole of some duration, they may assume various characteristics. Those that have been most frequently

FIGURE 1. *Lymphatic Drainage of the Skin of the Face.*

brought to our attention are an increase in size, an increase or decrease in pigmentation, and ulceration and bleeding. A few patients have described a sudden elevation followed by a central necrosis. These changes may occur throughout the lesion or in only a part of it (Case 25).

Once the tumors have assumed a malignant character, they may at any time produce widespread metastases. They may grow by direct extension, although this is unusual. Metastatic foci in the skin may occur locally or in close proximity to the primary focus, or may follow the main trunks in the skin lymphatics. By the latter route tumor cells may reach the regional nodes, or they may avoid the skin and travel solely through the deep lymphatic system to the same regional nodes. A third method of dissemination is by way of the blood stream, which may produce metastatic foci in the liver, lungs, brain, bones or skin distant from the primary lesion. This may be a primary or secondary phenomenon, that is from the primary tumor or from the regional nodes which have received tumor cells from the lymphatic system. There is no known way of predicting by which route or routes a given tumor will metastasize.

With the above facts in mind, we have attempted to determine the ideal method of treatment. There is no surgical method of combating blood-stream metastases, and if these have occurred before treatment, cure is impossible. The first step in adequate treatment is wide excision of the local lesion, care being taken not to traumatize the tumor, and to discard any instruments coming in contact with it during removal. A wide margin of apparently normal skin is important, and the tumor should be removed without regard to primary closure.

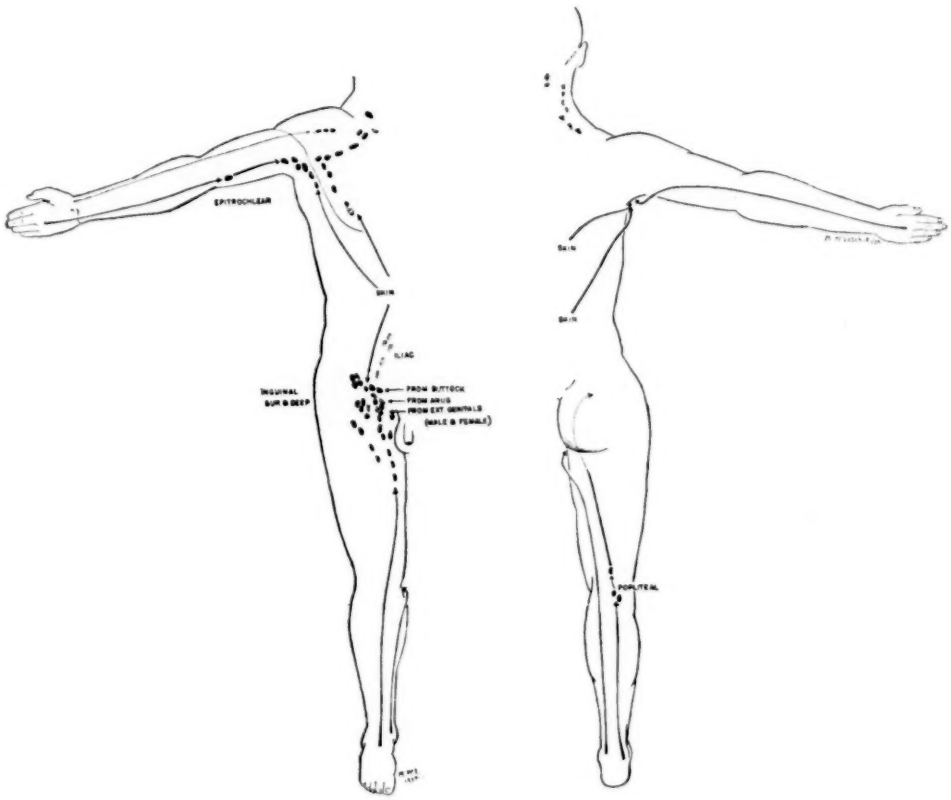

FIGURE 2. *Lymphatic Drainage of the Trunk and Extremities.*

The second procedure should be the thorough removal of the regional lymph nodes; these drainage areas are illustrated in Figures 1 and 2. Such dissections are major procedures; yet if they are performed properly and at the right time, the operative complications are slight. We do not subscribe to the doctrine that regional dissection should be deferred until stray tumor cells in the lymphatics have had time to migrate to the nodes. Therefore, if there is no ulceration or infection in the primary lesion, regional dissection may be performed at the time of the removal of the local tumor. If there is a possibility of infection, the regional dissection should be deferred for at least

two weeks or until the possibility of infection in the drainage area is past. By removal of the regional nodes the lymphatic routes have been cut off. This means that if there is to be a recurrence through lymphatic spread it will occur in the region between the primary site and the scar of the regional dissection. In this way the mutilating procedure of complete removal of skin lymphatics, which would be the ideal surgical treatment, is avoided. The lymphatics between the primary site and the regional drainage area may be removed when indicated by recurrent disease. This involves removal of the skin, subcutaneous tissue and superficial fascia down to the muscles (Case 7).

We realize that the treatment outlined above cannot be carried out in all cases. In many of our aged patients such an amount of surgery would not be tolerated, and the life expectancy would not justify anything more than an adequate local excision. The location of the lesion is another factor which hinders regional dissection. Malignant melanomas located directly above the sternum (Case 18) drain primarily to the nodes of the mediastinum and to those on both sides of the neck. Removal of the cervical nodes without the mediastinal nodes is ineffective.

As malignant melanomas of the extremities are the most suitable ones for excision and regional dissection, and as they represent over a third of the cases in our series, their records were closely analyzed in order to determine the efficacy of the proposed program. For lesions on the arm to be considered as having had an adequate regional dissection, an axillary one was required; for those on the leg, an inguinal and saphenous dissection from the lowest angle of Scarpa's triangle was necessary. Many of the cases with lesions on the extremities had local excision only, and although this group is small, it offers some comparison with those receiving the full treatment outlined above. Seventeen patients had local excision only, and 8 of these were dead within three years. Three cases were too recent to evaluate, and in 3 the patients were alive and free of disease two and a half, four and six years, respectively, after excision. Three records had no follow-up notes.

The cases that had the benefit of both local excision and regional dissection were divided into two groups: those with negative regional nodes and those with metastatic disease. In the latter group there were 24 cases. Seven of these patients are living; 3 of these have been operated on too recently to be considered as cures, 3 patients are living five years after treatment, and 1 who has recently had recurrent nodules removed from the scar of the saphenous dissection is alive two and a half years after primary treatment. Sixteen of the patients who died succumbed to distant or widespread metastases within two years. One patient lived for eleven years, only to die of probable brain metastases. Of the 13 patients who had no disease in the regional nodes, 5 were dead of recurrent disease within two years. There are 4 five-year cures, and 1 patient is free of disease after two years. Two patients were untraced.

In the group of cases which were considered operable when first seen, 16 patients died of distant metastases within a year after treatment. X-ray films of the chest, spine and pelvis were taken in most cases, and chest plates were taken routinely before operation. An exact note regarding the presence or absence of the liver edge was usually found, and although the data on melanuria were incomplete, the absence of melanin did not rule out distant metastatic disease.

Eleven patients with lesions on the extremities had either inoperable regional metastases or extensive distant lesions. All were dead within two years after treatment excepting 1 (Case 35) who showed spontaneous regression of the regional metastatic disease but died six years later of brain metastases. Three cases were lost, and 1 was not included in the group as the original saphenous dissection was not considered adequate.

There were 25 malignant melanomas of the orbit in our series. All but 3 of these were secondary cases, appearing in our clinics with metastatic lesions. In the 3 primary cases the patients are alive without disease nine and a half, six and two years, respectively, after treatment. None of the patients with secondary lesions survived.

Apparently a simple enucleation of the eye is adequate local treatment, for in only 2 cases was there a recurrence in the orbit. It is important to note that in but 1 case did we find direct metastases to the cervical nodes from a malignant melanoma in the uveal tract. There were 2 cases with widespread skin metastases where it was believed that secondary deposits from the skin tumors appeared in the regional nodes of the neck, axilla and inguinal region. Apparently no regional dissection is needed in malignant melanomas of this region.

There is indeed reason for the existing pessimism as to the permanent cure of malignant melanoma of the orbit. As explained previously, we did not have enough primary cases to enable us to draw any conclusions as to the number of cures during this period, for the patients were treated in eye clinics. We were impressed, however, by the number of patients returning with late metastases (Table 5). That tumors of this type metastasize later than any other type is common

knowledge. We have known of metastatic foci appearing eighteen and twenty-five years, respectively, after operation.

In this series there were late metastases to the liver in 10 cases, to the lungs in 9, to the brain in 3, to the bone in 2 and to the skin or subcutaneous tissues in 4.

Forty-one patients had lesions on some part of the face or neck. These patients fall into the group where local excision and regional dissection may be performed. However, we find that neck

TABLE 5. *Time Interval from Enucleation to Distant Metastasis in Cases with Malignant Melanoma of the Eye.*

TIME AFTER OPERATION	NO. OF CASES
Less than 1 year	3
1–2 years	4
2–3 years	5
3–5 years	2
5–6 years	1
6–7 years	1
7–9 years	1
9–10 years	2
No recurrence	3
Untraced or recent	3
Total	25

dissection was carried out in only 8 cases. Six patients had an adequate local excision, with positive nodes found on dissection of the neck. One is untraced; 3 died in six months, fourteen months and two years, respectively, and 1 case is recent. Two patients had the same operative procedure, but no disease was found in the nodes. These are comparatively recent cases, but both patients are free from disease, one for fourteen months and the other for seven. One patient had a secondary neck dissection but succumbed in four months.

Local excision alone was done in 17 cases. Three of these patients are untraced and 1 was well for ten years and then developed a recurrence in the neck. There are 5 patients living without disease, and 4 have died without recurrence; but none in either group has or had survived five years. Four patients died of early recurrence.

Five patients received x-ray treatment without benefit, and in 6 cases the disease was too far advanced for any treatment.

There are 2 five-year cures in this group of face and neck cases. Furthermore, only 7 patients have lived three years or more after receiving treatment. Malignant melanoma of the face and neck is obviously a serious condition and the prognosis is very poor.

For lesions on the trunk, as elsewhere, the ideal treatment includes a regional dissection, but only 6 patients received such therapy. The results here were poor; 2 postoperative deaths, 3 deaths within a year and 1 case untraced. There were 3 five-year cures by local excision alone. From the results in this small series there is little to be learned, but the probability is that a few of the patients who died of recurrent disease might have been saved if they had had the benefit of a regional dissection at the time of the primary treatment.

There were 12 cases of bone metastases from this type of tumor. The spine was involved in 3 cases, the skull in 2, the femur in 2, the ilium in 2 and multiple bones in 3. Plewes,[11] in a report of 97 cases from the Toronto General Hospital, found bone metastases in only 2 cases. Geschickter and Copeland[12] demonstrated bone metastases in only 3 of 169 cases. While the incidence of bone metastases is relatively low, even with our incomplete data, 12 cases were found in 174 cases studied.

The results of roentgen therapy were extremely poor. Twenty-two cases received sufficient therapy to determine their sensitivity. Of these, 2 patients (Cases 33 and 34) responded to treatment. One patient who received therapy to metastatic nodes showed good response but died in eight months of cerebral metastases. The other showed complete disappearance of a large mass in the groin and at least regression of lung metastases and was free of all symptoms two and a half years later. These results as a whole are in contrast to such reports as are found in the literature.[13]

The results of treatment are not very encouraging. Of the 90 patients who came to the hospital for primary treatment 50 are dead. There were only 13 five-year cures and in only 3 of these cases were there positive regional metastases. Of these 90 primary cases 21 were treated within the last five years. Six received palliative irradiation and 2 were untreated. This leaves 61 cases in which cure was attempted more than five years ago and 10 untraced. The percentage of cures in the primary group is 21.

The secondary cases include those in which local excision, cauterization, electrodesiccation or incision was done before entry, and those patients who had received adequate primary treatment elsewhere and were admitted for the treatment of late metastases. The results in this group are very discouraging. There are 2 patients, however, with regional metastases who have been cured for a five-year period. Two others are free from disease fourteen months and two and a half years after treatment, respectively. Two died of intercurrent disease three years and four years, respectively, after treatment.

There were 84 secondary cases in this series, but only 24 were treated with any possibility of cure. Three of these are recent. We thus had 2 five-year cures out of 21 possible cases, with 2 patients

untraced. The percentage of cures in this group was 9.

Of all cases, both primary and secondary, there were 3 five-year cures out of a possible 35 in the group with regional metastases. In the group without regional metastases, there were 12 five-year cures out of a possible 47. The curability of the operable cases in this series, then, was 9 per cent in cases with regional lymph-node involvement, and 25 in those without it.

CONCLUSIONS

The prognosis in malignant melanoma is very poor. However, a few patients can be cured by adequate surgery.

There is a high incidence of malignant melanomas on the lower extremities, and they represent more than half of the malignant tumors of the skin in this region.

Malignant melanomas arise in congenital pigmented nevi or as spontaneous primary growths. They rarely arise in pigmented, hairy nevi.

Trauma to a pre-existing lesion is probably a factor in stimulation of lawless growth. Cauterization or desiccation is dangerous.

Metastases may occur through the skin lymphatics or the deep lymphatics or via the blood stream.

Adequate surgical treatment includes wide local removal and thorough dissection of the regional nodes. We realize that the latter procedure is not always feasible.

In malignant melanomas of the eye, enucleation is adequate treatment.

Roentgen therapy is extremely unsatisfactory, but rarely there is benefit. It should be considered in inoperable cases.

Spontaneous regression may occur in the metastases.

While the prognosis is particularly poor in cases with regional lymph-node involvement, an occasional cure may be obtained. Patients should be given the benefit of adequate regional dissection.

Case Reports

ALIVE WITHOUT RECURRENCE

Case 1 (M. G. H. 268876). A 60-year-old woman was admitted to the Massachusetts General Hospital in 1925 with a pigmented lesion of the finger. Amputation of the finger and epitrochlear and axillary dissections were performed, with a positive report on the finger and a negative one on the nodes. She was recently examined and was found to be well. (Survival, 13½ years.)

Case 2 (private). A 47-year-old woman was admitted to the New England Deaconess Hospital in 1927. A black mole on her forearm had been removed 2 years before. She complained of a large mass in the axilla with pressure on the nerves of the arm. Dissection of the axilla was done with little expectation of anything more than relief. The pathologist reported melanotic sarcoma in the nodes. The patient has been examined regularly since then and is free from recurrence. (Survival, 10 years.)

Case 3 (M. G. H. 296322). A 41-year-old man was admitted to the Massachusetts General Hospital in 1929. He complained of a tumor of the inner aspect of the leg of 4 years' duration. There had been gradual growth and ulceration and the tumor had become pedunculated. Excision and saphenous dissection were done. The primary growth showed melanotic sarcoma, but the nodes were negative. A letter from the patient in 1938 stated that he was in good condition except for stomach symptoms which he had had for several years. There has been no examination, but he is probably free of disease. (Survival, 9½ years.)

Case 4 (private). A 24-year-old man complained of failing vision. A pigmented tumor of the iris was found and enucleation was carried out by an ophthalmologist. The pathologist reported the tumor to be a malignant melanoma. The patient was free from disease 9½ years later.

Case 5 (M. G. H. 292449). A 25-year-old woman was admitted to the Massachusetts General Hospital in 1928. A nodule on the forearm appeared 2 years previously. Three months before entry a node was felt in the epitrochlear region and 1 month later one in the axilla. The tumor was excised together with the epitrochlear and axillary nodes, all of which showed malignant melanoma. The patient was examined and found free of disease in 1937. (Survival, 9 years.)

Case 6 (P. H. 4252). A 63-year-old housewife entered the Pondville Hospital in February, 1932. She had had a mole on her back for 6 years. Three weeks before admission it began to grow and the skin about it became inflamed. Local examination showed an elevated crusted lesion of the skin overlying the lumbar spine. Wide excision of the lesion was done without regional dissection. The pathological report was malignant melanoma. Since then she has been well and free of disease. (Survival, 6½ years.)

Case 7 (P. H. 4272). A 51-year-old male entered the Pondville Hospital in February, 1932. Nine months before admission he had struck his heel and broken the skin. His shoe had kept this lesion irritated and a few weeks later his physician had cauterized it with silver nitrate. Six weeks before admission he had noticed a swelling in the right groin. An indurated growth 3 cm. in diameter was found on the inner aspect of the right heel. There was a freely movable mass of nodes in the right saphenous

FIGURE 3. *Case 7.*

The recurrent nodules in the skin have been removed with the skin, subcutaneous tissue and fascia, and the defect grafted.

region. A wide excision of the local lesion together with a saphenous and inguinal dissection was done. The pathological report was amelanotic malignant melanoma with metastases to regional lymph nodes. Four months later the patient noticed nodules on the inner aspect of the leg and entered the hospital for excision of the skin of the inner side of the leg. This required multiple skin grafts (Fig. 3). The nodules were amelanotic malignant melanomas. The excised skin measured 51 by 8 by 1.5 cm. He later had a recurrent nodule removed from the upper end of the scar but was free of disease 6½ years after treatment.

Case 8 (P. H. 5309). A 52-year-old housewife was admitted to the Pondville Hospital in August, 1932. She had noticed poor vision in the left eye for a number of months. In the upper inner quadrant of the left eye the iris was bulging forward. Posterior to this there was an apparent hemorrhagic pigmented area of new growth. Enucleation of the eye was performed and a diagnosis of malignant melanoma of the iris was made. She has since been free of disease. (Survival, 6 years.)

Case 9 (M. G. H., Baker, 9358). A 41-year-old woman was admitted to the Massachusetts General Hospital in March, 1933, for an abdominal operation. She called our attention to a pigmented mole on her back. This had been present for several years and had shown no recent change. It was excised with a wide margin and showed melanotic sarcoma. No regional dissection was done, as there were no palpable nodes in either axilla. The patient was apparently free of disease in September, 1938. (Survival, 5½ years.)

Case 10 (M. G. H. 330480). A 25-year-old woman was admitted to the Massachusetts General Hospital in 1933. A mole on the dorsum of the hand, which had been traumatized frequently, grew rapidly and spread to the axillary nodes. Amputation at mid-forearm and dissection of the axilla were done. The pathologist reported a very rapidly growing melanotic sarcoma of the amelanotic type. A letter from the patient in 1938 stated that she was well. (Survival, 5 years.)

Case 11 (M. G. H. 337411). A 62-year-old woman was admitted to the Massachusetts General Hospital in 1934 with a tumor over the malar prominence of 3 years' duration. There had been a gradual increase in size, but there were no palpable nodes. Excision with a plastic closure was done, but there was no regional dissection. The pathological report was melanotic sarcoma. A letter from the patient in 1938 stated that she was well. (Survival, 4½ years.)

Case 12 (P. H. 4412). A 34-year-old man was admitted to the Pondville Hospital in March, 1932, with a growth on his leg of 12 years' duration. There had been rapid growth for 1 year without antecedent trauma. The lesion was elevated, non-ulcerated, and slightly bluish. Only wide local excision was performed. Since then, he has been well. (Survival, 4½ years.)

Case 13 (M. G. H. 339669). A 63-year-old woman was admitted to the Massachusetts General Hospital in 1934 with a lesion on the sole of the foot. This had been desiccated in the Out Patient Department 2 weeks previously but had promptly recurred. A wide excision was done without regional dissection, with a report of melanotic sarcoma. A letter from the patient 4 years later stated that she was well. (Survival, 4 years.)

Case 14 (M. G. H. 307524). A 43-year-old woman was admitted to the Massachusetts General Hospital in 1930 with a lesion 2.5 by 2.5 cm. on the chest wall. This was excised and reported to be a malignant melanoma. Five years later there was a local recurrence and a second excision was done, with a positive pathological examination. No regional dissection was done. The patient was free from disease in 1938. (Survival, 8 years.)

FIGURE 4. *Malignant Melanoma of the Foot. Partial amputation was performed. Dissection was advised but refused. The case is now untraced.*

Case 15 (M. G. H. 85397). A 27-year-old woman was admitted to the Massachusetts General Hospital in 1935 with a pigmented lesion on the buttock. A wide excision with a saphenous and groin dissection was done. Pathologically this was a melanotic sarcoma with positive nodes. There was a recurrence in the scar 2½ years later but no further trouble in the regional drainage area. The second pathological report was positive.

Case 16 (P. H. 10536). A 64-year-old housewife entered the Pondville Hospital in March, 1936. She had been blind in the left eye for 13 years. An enucleation of the eye had been done at an outside hospital 1 month before admission. Examination showed a mass of red tissue covered with mucous membrane protruding between the eyelids. An exenteration of the left orbit was performed and a diagnosis of malignant melanoma was made. The patient was living and well 2½ years after operation.

Case 17 (P. H. 9797). A 15-year-old girl entered the Pondville Hospital in August, 1935. A birthmark on the left cheek had grown larger and blacker since birth. A pigmented nevus 3 mm. in diameter was removed, and the pathological report was that of malignant melanoma. No neck dissection was done. There was no evidence of recurrence 2 years after treatment.

Case 18 (P. H. 11116). A 52-year-old woman was admitted to the Pondville Hospital in July, 1936. She had had a mole on the neck for 5 years. Recently it had grown larger and become black. Examination showed an irregularly shaped, deeply pigmented nevus 1 cm. in diameter just above the clavicle and just to the right of the

midline of the neck. Wide excision was performed, but no regional dissection, as one of the primary lymphatic drainage areas was to the nodes of the mediastinum. The patient was well and free of disease 2 years after excision.

Case 19 (P. H. 11745). A 42-year-old housewife entered the Pondville Hospital in November, 1936. For 10 years she had noticed a firm swelling over the distal joint of the left great toe. The swelling gradually increased. On the

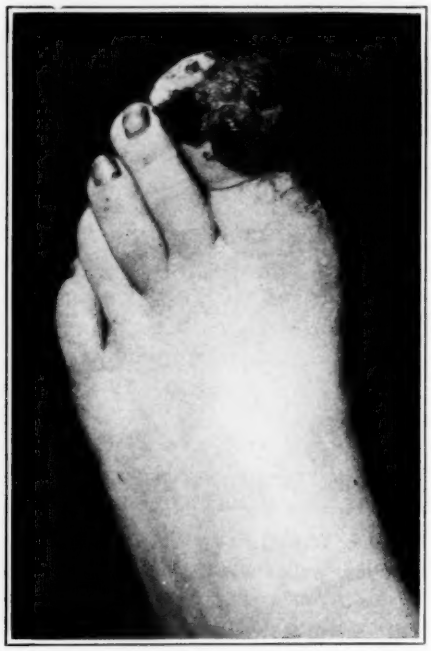

FIGURE 5. *Malignant Melanoma of the Great Toe. This patient died ten months after operation.*

dorsum of the left great toe, a large, lobulated, slightly purplish, firm tumor was found. The tumor was found to be encapsulated and was excised locally. No saphenous or inguinal dissection was done. The diagnosis was that of malignant melanoma. The patient was well and free of disease 22 months after the local excision.

Case 20 (P. H. 8521). A 32-year-old housewife entered the Pondville Hospital in July, 1937. A birthmark just below the right ear had started to increase in size 7 weeks before admission. A wide excision of the lesion was done, followed by neck dissection 3 months later, postponed because of a low-grade dermatitis. The pathological report was malignant melanoma without metastases to the regional lymph nodes. Soft nodes overlying the sternomastoid muscle were removed in 1938. These were negative. The patient was well and free of disease 14 months after treatment.

Case 21 (P. H. 12606). A 36-year-old woman was admitted to the Pondville Hospital in June, 1937. Two years before admission she had had a mole removed by freezing. The mole had been present only a few months but was black and ulcerated. A month after removal a small lump was felt in the region of the trapezius muscle. This had gradually increased in size. Examination showed a 7-cm. mass in the subcutaneous tissue in the region of the trapezius muscle. There was no recurrence locally, and the mass was removed together with a dissection of the posterior carotid triangle. The pathological report was malignant melanoma with metastases to the cervical lymph nodes. The patient was well and free of disease 14 months after operation.

Case 22 (P. H. 12780). A 66-year-old man was admitted to the Pondville Hospital in July, 1937. He had had a smooth, slowly growing mole on the left cheek for 15 years. He had noticed that this was becoming thicker. Examination showed an irregular, black, movable lesion in the center of the left cheek. In the central portion of the lesion there was a warty, raised, thickened area. A wide local excision with plastic closure was done. The diagnosis was that of malignant melanoma. No neck dissection was done. The patient was well and free of disease 13 months after operation.

Case 23 (P. H. 11908). A 49-year-old housewife was admitted to the Pondville Hospital in January, 1937. Six months before admission she noticed a lump near her rectum. Four weeks previously part of the tumor was removed and the pathological report was melanotic sarcoma. A radical excision of the scar was performed and only one focus of tumor cells was found in the specimen. Five months later a node was found in the right groin, and inguinal and saphenous dissections were done. Nodes were positive for metastatic malignant melanoma. The patient was well and free of disease 18 months after the primary excision and 12 months after the inguinal and saphenous dissections.

Case 24 (M. G. H. 100780). A 33-year-old woman was admitted to the Massachusetts General Hospital in December, 1937, with a black mole on the neck below the ear. A wide local excision was performed and the mole was found to be a malignant melanoma. A radical neck dissection was performed 1 month later but the nodes were negative for tumor. One lymph node which lay above the clavicle appeared black grossly, but anthracosis was found microscopically. The patient was apparently free of disease in August, 1938. (Survival, 9 months.)

Case 25 (M. G. H., Baker, 107537). A 41-year-old woman was admitted to the Massachusetts General Hospital in February, 1938. A pigmented mole on the instep which had been present since birth had changed in appearance and had become sore over a period of 8 months. Examination showed a flat, brown lesion 2.5 by 2 cm., the center of which was raised, indurated and pinkish. Clinically the center appeared malignant and the periphery benign. This proved to be the case when the lesion was excised. A dissection of the saphenous and inguinal regions was done 6 days later and negative nodes were found. There has been no recurrence in the 7½ months since operation.

Case 26 (W. S., Cancer Section, 258). A 57-year-old woman was admitted to the Westfield Sanatorium in March, 1938, with a lesion on the cornea. One year before a similar lesion had been treated by radium, apparently with benefit. Biopsy showed a melanotic sarcoma of the amelanotic type. The eye was enucleated. The patient was apparently well 6 months later.

Case 27 (M. G. H., Phillips House, 106785). A 77-year-old man was admitted to the Massachusetts General Hospital in January, 1938, with a black mole of 5 years' duration on his back but with recent enlargement. Several other flatter lesions with less pigment had been treated by

a dermatologist with radium over a period of 2 years. He had advised against treatment of any sort for the black mole. However, excision was done and a positive report obtained. This patient has been followed for about 6 months and is apparently well.

Case 28 (W. S., Cancer Section, 449). A 22-year-old woman was admitted to the Westfield Sanatorium in May, 1938, with a lesion on the lower eyelid of 1 month's duration. It had been treated by an electric needle before entry. Excision was done at once, and the report was amelanotic sarcoma. Because the removal did not appear to be wide enough for this type of tumor, a wider removal was done by excising half the eyelid and reconstructing a new eyelid. The second microscopic examination showed more of the same type of tumor. This patient has been followed for but 2 months after operation.

Case 29 (M. G. H., Baker, 131786). A 68-year-old man was seen in consultation in May, 1938. He said that a pigmented tumor had been present on his face for 4 years. Two years previously he had been treated with an electric needle and the tumor had partially disappeared. Six weeks before examination a node had appeared in his neck. Operation was advised and accepted. The primary tumor together with the skin between it and the node was removed, and a radical dissection of the neck was done. Pathologically this was melanotic sarcoma with metastasis in a node adherent to the parotid gland. This is a very recent case and has been followed but 2 months.

DIED WITHOUT RECURRENCE AFTER FIVE YEARS

Case 30 (M. G. H. 200519). A 60-year-old man was admitted to the Massachusetts General Hospital in 1915. A mole on the leg of 10 years' duration broke down and crusted over. A wide excision with saphenous and inguinal dissection was done. The primary lesion showed melanotic sarcoma, but the nodes were negative. The patient was found free of disease 2 years later. He died of other causes in 1936. (Survival, 21 years.)

Case 31 (M. G. H. 295000). A 74-year-old man was admitted to the Massachusetts General Hospital in 1928 with a nodule on his back measuring 5 by 6 cm. Excision was done and the tumor was found to be a melanotic sarcoma. There was an immediate recurrence and a second excision, again with a positive report. The patient died without recurrence 5 years later.

Case 32 (M. G. H. 268397). A 75-year-old woman was admitted to the Massachusetts General Hospital in 1925 with a 2-by-2-cm. pigmented mole on the cheek. The lesion was excised and reported to be a malignant melanoma. The patient died 10 years later without recurrence.

TREATMENT BY X-RAY

Case 33 (M. G. H., Baker, 15387). A 67-year-old man was seen in consultation in June, 1936. A short time previously he had been operated on for a large tumor in the femoral region. The tumor proved to be unremovable. It was a rapidly growing amelanotic type of malignant melanoma. When examined he had a large mass in the inguinal and saphenous region and the tumor could be felt in the pelvis. There was some swelling of the leg and a lymph sinus in the wound. X-ray treatment was given to the mass as a palliative measure. A total of 4000 r (200 kilovolts) was given through a 15-by-15-cm. field to the mass in the pelvis, groin and saphenous region, the treatment being spaced over 2 weeks. There was immediate regression and disappearance of the tumor. Three months later the patient complained of a severe cough. A chest plate showed probable pulmonary metastases. He was given 800 r both to the front and the back of the chest. His cough cleared up at once. During the 2½ years since treatment he has had no recurrence of the tumor in the groin. Chest plates show no changes. He is free from symptoms and is able to continue at his work.*

Case 34 (P. H. 13063). A 64-year-old man was admitted to the Phillips House in September, 1937. He had had a mole on his back in the region of the scapula for several years. Ten months before admission this became sore and was excised by his doctor. Three weeks later his neck began to enlarge, and nodes were later noted in his axilla. Two months previously he had noted nodes in his groin. Examination showed a diffuse, hard mass above the clavicle, and a baseball-sized mass in the axilla which was adherent to the deep structures. A chest plate showed no evidence of metastatic disease. The patient received 1200 r to the right axilla, right supraclavicular area and scrotum in October, 1937. The masses in the axilla and supraclavicular areas showed regression, and in December, 1937, he received an additional 600 r to each of these areas. In March, 1938, further radiation, 1200 r to the axilla and 600 r to the groin, was given.

CASE WITH SPONTANEOUS REGRESSION

Case 35 (P. H. 1064). A 43-year-old man was admitted to the Pondville Hospital in January, 1929. Two years before admission he had had the 5th toe removed for a "black tumor." An inguinal dissection had been done. Several months before admission he had had erysipelas of the lower leg, followed by the appearance of black nodules. Multiple nodules were found over the lower leg, and there was a fixed mass in the groin but no nodules above the operative area. The case was considered inoperable and no treatment was given. A year later the masses in the thigh began to disappear, leaving only the pigment. The masses in the thigh finally disappeared, and the patient died of probable brain metastases 6 years after his first admission.

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Discussion

Dr. George F. Dwinell, Manchester, New Hampshire: This is a peculiarly difficult subject to discuss because we do not see very many of these cases. One hundred and fifty-eight cases is an unusual number to report. I have

*This patient died in March, 1939, following operation for a strangulated hernia. An autopsy showed no trace of his original tumor and no metastases.

looked up several series, and 50, I believe, was the next highest number that anyone reported; and I think this series shows results about as good as are shown elsewhere. Twenty per cent is a high incidence of five-year cures.

We have no large series in Manchester, and I have not attempted, nor has Dr. Wilkins, to look up all our cases, but he has asked me to bring before the society that following a definite routine which he has adopted, and which I have used, we have 3 outstanding cures. One is that of a patient with a lesion on the face who has had no recurrence for over ten years. (The mortality is very high in face cases.) The other 2 had lesions of the extremities. One of the latter came to me three years ago, and I did not think of the possibility of melanoma. I took a biopsy, thinking the case was probably one of fibrosarcoma, and that was the report which the pathologist made on the little piece that I sent him; but later I dissected the nodes of the groin and found a typical melanosarcoma.

The point which Dr. Wilkins wishes me to bring out is that perhaps we have been a little bit lax in our treatment. These cases must be handled perhaps a little more radically than Dr. Daland and Dr. Holmes brought out. All our modalities should be used. The method we use consists, in the first place, of large doses of buried radium, not in the lesion but surrounding it, and also radium on the surface. By large doses I mean 1800 mg. hours — 200 mg. of needles and tubes buried around the lesion for nine or ten hours. This is to be followed in about three weeks, at which time the height of the radium reaction is supposed to occur, by complete excision. This means wide excision, at least 1 cm. away from the lesion, extreme care being taken not to touch the lesion. Furthermore — and Dr. Daland does not agree in this — we believe that dissection of the nodes should be deferred for two or three weeks. As you know, melanoma gives a very peculiar pathological picture. It does not travel the way that ordinary cancer cells travel. It travels in the skin, usually just under the surface. Our idea was to allow these cells to reach the lymph nodes and be caught there before dissection. We follow up the regional lymph-node dissection with a thorough course of deep x-ray therapy.

After this I do not believe that any more can be done. The 3 cases previously mentioned have been treated by this method, and have remained free from recurrences for ten, three and three years respectively.

This is the main point that Dr. Wilkins wished me to bring out: that we ought to do a little more than we have in the past for these cases, and should not merely say that radium has no effect. The effect of our initial radiation, I think, is purely and simply one of fibrosis. All these tumors practically disappear. I do not believe that the radium kills the growth, but that loss of nutrition does; however the method is worth trying in the way that Dr. Wilkins suggests.

Dr. Edward H. Risley, Waterville, Maine: I should like to emphasize a point which I have only recently learned from a bitter experience. I think most of us have always assumed that a melanotic tumor could be recognized by the melanosis in the tumor itself. I find that this is not so. There are many small tumors which appear on the forearm, and on other extremities, which look like small fibromas or some other form of tumor, but which on removal turn out to be malignant melanomas.

I operated on a woman of thirty-six for what felt like a fibroma on the forearm. I excised it with the endotherm and found that she had no diseased nodes in the axilla. The pathological report was malignant melanoma. In July the patient had a well-developed metastasis in the axilla, and the following December she died of a brain metastasis.

I have seen one other case of a similar nature, and I believe we should not do biopsies on these tumors of the extremities; we should excise them and then get our diagnosis, rather than depend on a biopsy, which is many times not a safe procedure.

Dr. Grantley W. Taylor, Boston: I should like to ask whether Dr. Daland and Dr. Holmes have a program of therapy for pigmented nevi.

Dr. Daland (closing): We found no case with a history of hair in the nevi, nor have we found any described in the literature. There was only 1 case with a lesion on the scalp. Adair has quoted a study of 250 patients who had an average of twenty pigmented nevi per patient. We may talk about removing all pigmented nevi, twenty from each of 250 patients, but it cannot be done. There are not enough surgeons to do it, but if there is a pigmented nevus in a place where it is subject to any type of irritation, it should be excised and not be treated by desiccation.

I am glad that Dr. Risley brought out his point. Fully half these cases show no pigment, but I want to emphasize again that most malignant tumors of the skin of the leg are carcinomas or malignant melanomas, and should be excised. Let the pathologist see the tumor after it has been removed in its entirety.

THE TREATMENT OF CHRONIC PRURITUS VULVAE WITH LOCAL APPLICATIONS OF ESTROGEN*

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THERE are few conditions which confront the physician that are more resistant to treatment than is chronic pruritus vulvae associated with leukoplakic and kraurotic changes of the labial skin. The intolerable itching frequently assumes such proportions that, despite attempts at palliation with antipruritic salves or lotions, alcoholic injections and exposure to x-rays, there is continual psychic trauma and loss of sleep, resulting in general physical debility. These cases, to which no demonstrable cause has as yet been assigned, may be classified as an essential type as differentiated from the commoner type in which the skin changes are not a prominent feature and the causative factor or factors are apparent and remediable.

Cases of pruritus vulvae other than the essential type are the result of local irritation from chronic cervicitis, trichomonas vaginalis vaginitis, infection with the epidermophyton, diabetic or infected urine or obesity with gross uncleanliness. This type rarely continues to the chronic state, because adequate treatment directed to the removal of these respective causative factors relieves the patient of her symptoms.

It is the purpose of this paper to present a hypothesis for the pathogenesis of the essential type of chronic pruritus vulvae with leukoplakic and kraurotic changes, and to report the results following treatment of a group of 4 cases with locally applied estrogenic substances. The collective term "leukoplakic vulvitis" will be used in referring to these cases.

Although the results following denervation of the affected area offer some encouragement in the control of this condition, the permanency of relief is not established and the procedure is contraindicated in some 40 per cent of cases (Learmonth et al.,[1] Montgomery et al.[2] and Usher and Campbell[3]). A still more radical approach to therapy is vulvectomy, which is not infrequently followed by considerable local discomfort and frequent recurrences. Vulvectomy has its usefulness in cases of malignancy, or in cases where malignant changes are anticipated.

Disturbance in ovarian function has for many years been considered an etiologic factor in leukoplakic vulvitis, but it has not been proved an important one. The most probable reason for this may well be the fact that substitutive treatment with estrogenic hormones has proved unsatisfactory. In most cases, however, this treatment has been started many years after pruritus, the first and frequently the only symptom, was noted by the patient.

There are many facts which support the theory of decreased ovarian function as a contributing causative factor. The statistics of reported series show that the average age of occurrence of leukoplakic vulvitis is 52 years, an age well past the time when the ovary begins to fail in function. The region of the vulva in women may possibly be considered as a true "sexual skin." In the monkey and in certain of the great apes that skin about the vulvar and gluteal regions manifests marked vascular changes coincident with the follicular phase of the menstrual cycle. This great reddening and edema seen in young animals at the time of ovulation has been shown to be due to the unopposed action of estrogen (Allen[4]). Although no such sexual skin has been demonstrated in the human being, it appears likely that a homologous area may exist and be specifically affected by estrogen. Such a concept receives support from the observation that considerable atrophy of the vulva normally follows castration and the menopause. It is quite conceivable that this physiologic atrophy may render the cutaneous area more liable to pathologic changes incident to any damaging influence.

There is significant histological evidence that in the cases under consideration there is a progressive disintegration of the elastic fibers in the corium. This loss of elasticity, together with the epithelial changes, results in a thickened and rigid skin which cracks easily, opening portals for subepithelial infection. Several factors may then irritate the nerve endings sufficiently to produce a sensation of itching. Once pruritus is established, the constant trauma of scratching inevitably leads to excoriation of the skin, and eventually to a chronic pathologic state with hyperplasia and sclerosis in variable proportions.

Parenteral estrogen therapy has offered relief to only a small percentage of women suffering from leukoplakic vulvitis. As stated above, the treatment has in most cases been instituted many years after the onset of symptoms, conceivably

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after the local changes have been established for a long time.

It appears that parenterally administered estrogen, even in large amounts, is less efficacious in local conditions than smaller amounts locally applied. This has been shown to be true in treating Neisserian vaginitis in children and young girls before puberty, and senile vaginitis at or after the menopause. Lyons and Templeton[5] have shown that locally applied estrogen is two hundred times as effective as parenteral estrogen on the vagina of a rat. In view of the rapid destruction of estrogen in the organism (Dingemanse and Laqueur[6]), it seems reasonable to expect that constant and continuous local action of estrogen can be better attained by frequent local applications. That estrogen is readily absorbed when applied to the human skin has been shown by Salmon.[7] Zondek,[8] and subsequently Klaften[9] and Reifferscheid,[10] reported successful therapy of pruritus vulvae by local percutaneous application of estrogen combined with large doses parenterally by injection. While this investigation was in progress it was noted that these earlier investigators[8, 9, 10] had used the estrogenic material in the form of a salve. Following their suggestion a lanolin base was used in the later treatment of the cases reported below. This has the advantage of greater ease of application and has proved just as efficient a vehicle as sesame oil. Local application has therefore been employed in the therapy of the cases reported below.

METHOD

The vulva and epipubic and perineal skin are thoroughly cleaned and dried. Estradiol* in sesame oil (0.5 mg. per cubic centimeter) is thoroughly massaged into the skin of the affected area. One cubic centimeter (60,000 international units) is used for each inunction at two- to six-day intervals. The patients are instructed to use no additional treatment. After the pruritus is controlled, smaller amounts, in the form of a salve with a lanolin base, are applied daily by the patient.

Case Reports

Case 1. A. D., a 44-year-old woman, who had had five children, was seen on June 24, 1938. A persistent, severe pruritus vulvae of 2 years' duration had not been influenced by any of various antipruritic ointments or lotions. There was no associated leukorrhea, glycosuria or infected urine. In August, 1935, the intrauterine application of 1200 mg. hr. of radium for functional bleeding was followed by amenorrhea until August, 1936, when there was another application of 1200 mg. hr. of radium for recrudescence of bleeding. There had been no further bleeding.

Physical examination showed extensive whitening of the skin of the labia majora with markedly thickened plaques of leukoplakia extending to the epipubic region. The labia minora were atrophic, and the vulvar orifice was narrowed. There were several excoriations. The uterus, cervix and vagina were atrophic.

An inunction of 60,000 units was applied on the first day. In 2 days, considerable relief was noted, and complete relief followed the application of 255,000 units over a 16-day period. Treatment was discontinued in order to determine the permanency of relief. No bleeding occurred after the withdrawal of estrin. After 6 weeks of complete relief there was a recurrence of the pruritus. One inunction of 60,000 units on August 26 brought about relief. The patient was then given a salve with a lanolin base containing 20,000 units of Estradiol per gram. By applying a small amount every evening she has remained symptom-free for 3 months, having used 20 gm. of the ointment.

Case 2. E. G., a 72-year-old woman, who had had one child, was seen on July 1, 1938. The menopause had occurred 22 years previously. Persistent pruritus vulvae of several years' duration had been associated for the previous 2 years with marked incontinence of urine. On April 5, 1938, a plastic repair was performed, with only partial relief of the incontinence and no influence on the pruritus.

Physical examination showed extensive involvement of the labia majora by multiple plaques of white, thickened epithelium. This extended to the thighs and the epipubic and perianal regions. The labia minora were replaced by thickened, white, shiny skin, and there was marked narrowing of the vulvar orifice. The entire area was moist, and there were many deep excoriations with secondary infection. The cervix and uterus were atrophic.

Six applications of approximately 60,000 units each, over 4 weeks, resulted in considerable but not complete subjective relief. However, in spite of the complaint of continued pruritus the excoriations incident to the long-practiced scratching disappeared. Inasmuch as the incontinence was not corrected, it appears that in this case the irritation from the urine played a part in the production of the pruritus. However, with daily applications of small amounts of a lanolin salve containing 60,000 units of Estradiol per gram the patient has had sufficient relief to allow uninterrupted sleep.

Case 3. J. K., a 35-year-old woman, who had had two children, was seen on July 25, 1938. Persistent pruritus vulvae of 3 years' duration was associated with vaginal discharge due to a chronic cervicitis with polyposis of the endocervical canal. Catamenia were regular and normal. Excision of the polyps and cauterization of the canal with a post-cautery in April, 1938, was followed by a diminution but not complete disappearance of the discharge. There was, however, no relief from the pruritus, and treatment with antipruritic salves and lotions was without effect.

Physical examination showed marked thickening of the vulva and of the perineal skin extending to the epipubic and perianal regions. The skin was grayish, and there were many excoriations. The cervix was well healed, and the canal smooth. There was, however, some purulent discharge from the endocervix at the internal os. The uterus and adnexa were normal.

*The estrogenic material and the lanolin salve were prepared and furnished through the courtesy of Dr. Max Gilbert of the Schering Corporation, Bloomfield, New Jersey.

Three applications of 60,000 units of Estradiol in oil, at weekly intervals, followed by five applications of 30,000 units at 3-day intervals, resulted in marked though not complete subjective relief. The attacks, however, were of short duration as opposed to attacks before treatment lasting for hours. The vulvar skin appeared more normal and was less thickened and there were no excoriations.

The patient was given a lanolin salve containing 60,000 units of Estradiol per gram. By applying a small amount every evening, the patient has since experienced complete relief from the pruritus.

Case 4. L. N., a 44-year-old woman, who had had two children, was seen on August 12, 1938. There was persistent though mild pruritus vulvae of 16 years' duration, associated with vaginal discharge and chronic cervicitis with polyposis of the endocervical canal. Catamenia were regular and normal. Excision of the polyps and cauterization of the cervical canal with a post-cautery were followed by a disappearance of the discharge. Relief of the pruritus was then noted except for an area in the epipubic region.

Physical examination showed the vulva to be normal in appearance except anteriorly in the epipubic region, where the skin was white, markedly thickened and excoriated. The cervix was well epithelialized, the canal smooth and there was no discharge. The uterus and adnexa were normal.

Over a 31-day period, 220,000 units of Estradiol in oil was applied to the affected area in daily divided doses. This was followed by relief of the pruritus and disappearance of the excoriations. By daily using a small amount of lanolin salve containing 60,000 units of Estradiol per gram, the patient has remained symptom-free. She was last seen, October 17, when the skin of the epipubic area was normal in color and texture.

It is apparent from these reports that the local application of a strong solution of estrogen has effectively remedied both the subjective symptoms and objective signs of pruritus vulvae. It is significant in this investigation that the psychological factors have been eliminated, in that objective criteria have been used in measuring the degree of relief. The patient's subjective sensations did not influence the objective sign of pruritus, namely the inevitable excoriations of the skin. The disappearance of these lesions can be accepted as evidence that the symptom had been relieved, if not completely cured. This objective evidence of relief was found in all the cases cited.

The noted effects can scarcely be ascribed to the oil, because all cases had been treated with emolient salves without any effect, and one patient (Case 2) experienced no relief of pruritus with daily inunctions of oil over a trial period of seven days. It was also observed that the relief obtained with a preparation containing 20,000 units of Estradiol per cubic centimeter of oil was very transient as compared with that obtained by using the more potent preparation (60,000 units).

CONCLUSIONS

A hypothesis for the pathogenesis of leukoplakic vulvitis on an endocrinological basis is presented.

A simple method of treating leukoplakic vulvitis by local application of estrogen is offered. It is recommended because continuous local action simulating normal physiological conditions may conceivably be attained by frequent inunctions by the patient.

The results obtained in treating a small group of 4 cases justify further investigations on selected cases to determine more definitely the doses required and the permanency of relief.

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AN OUTBREAK OF INFECTIOUS DIARRHEA AMONG NEWBORN INFANTS

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DURING November, 1937, an extremely disastrous epidemic of infectious diarrhea broke out among a group of newborn infants on the Pediatric Service of the Worcester Memorial Hospital. The clinical features and pathologic findings closely resembled those described by Rice and others[1] in their report on sixteen outbreaks of infectious diarrhea occurring in the nurseries of eleven hospitals in New York City. We have reason to believe that our cases arose directly following the admission to the ward of an infant who had been discharged six days before from a hospital in New York City.

By overcoming the natural reluctance of any one hospital to report its disastrous results, Dr. Rice, who is Health Commissioner of New York City, and his collaborators are to be highly commended for correlating and reporting the experiences of these hospitals. They contend that the cases of diarrhea covered in their report constitute a distinct disease entity, basing their argument on the following grounds: exact similarity of clinical picture; specificity of age group, the occurrence being exclusively among newborn infants; uniformly high morbidity and mortality rates (46 per cent mortality among 505 infants); highly contagious nature of the infection; inability to identify the causative organisms by ordinary bacteriological measures; and extremely mild pathologic findings referable to the gastrointestinal tract, associated, however, with secondary pyogenic infections. The cases here reported fulfill these criteria exactly.

We were extremely unfortunate in the group of infants that were housed on our Pediatric Service when the epidemic developed. As the highly infectious nature of this diarrhea became apparent, we placed the babies under increasingly strict isolation precautions, until finally each surviving infant was in a room by itself under entirely separate nursing care. Through prompt removal from the ward, two postoperative cases of hypertrophic pyloric stenosis were protected from infection. It is important to enumerate the facts concerning the babies who acquired the infection:

Case 1. D., a girl, was admitted to the hospital November 10, 1937, when 1 month old; the birth weight had been 7 lb. One week previously she had been discharged from a New York City hospital, where she had been exposed to a case of diarrhea. On admission she weighed 5 lb., 15 oz.; she was vomiting and having frequent loose movements, and appeared extremely toxic.

Case 2. P., a premature boy, was born outside the hospital and brought immediately to it; the birth weight had been 3 lb., 3 oz. At 1 month the weight had increased to 4 lb., 7 oz. The diarrhea started November 11. Several temperature reactions occurring previously were thought to be due to instability of his temperature-control mechanism.

Case 3. G1, a girl, was one of twins born in the hospital; the birth weight had been 4 lb., 2 oz. This infant offered considerable difficulty in taking her feedings, and on the 20th postpartum day (November 11), when she began to vomit and have loose, green stools, she weighed 4 lb., 5 oz.

Case 4. G2, a twin sister of Case 3, had weighed 4 lb., 4 oz., at birth. This infant also presented difficulty in feeding, and on the 21st postpartum day (November 12), when diarrhea began, she weighed 4 lb., 12 oz.

Case 5. B, a boy, was born during the course of pneumonia of his mother; the birth weight had been 7 lb., 12 oz. When 1 week old, he was weaned and placed on a formula; at this time the weight was 6 lb., 14 oz. The following morning (November 16) six or eight loose, greenish-yellow movements occurred.

Case 6. F., a boy, was referred, when 6 days old, from an out-of-town hospital for correction of a complete cleft palate and left harelip; the birth weight had been about 6 lb. On admission his weight was 5 lb., 10 oz. Because of his handicap, considerable difficulty was experienced in feeding, and at the onset of the diarrhea on November 18, when he was 21 days old, he weighed 6 lb., 2 oz.

Table 1 presents a summary of the clinical experiences with these 6 infants in course of their infectious diarrhea.

The mortality of this series is extremely high, the highest, in fact, of any of the reported series of cases. However, it is not inconsistent with an infection which extracted a case mortality of 46 per cent from the average run of healthy newborn infants who acquired the disease. Five of the 6 babies who acquired the disease succumbed to it. As appears from the above description, it would be hard to select a group of babies constituting a less favorable risk than those who acquired this infection in our ward. The infants were all within the age period which appears peculiarly susceptible to this disease. Although born of a mother who went into labor and delivered during the active course of pneumonia, Case 5, the sole survivor,

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was the only apparently normal infant in the group.

On the second or third day of their illness, when they were passing frequent loose or watery movements, the babies began to vomit. So far as we could see, it made little difference whether tap water, cereal water, weak tea or any of the various formulas was fed, even though given in small quantities and in great dilution. The babies at this stage vomited everything taken. By dint of repeated administration of 5 per cent glucose in saline and frequent injections of whole blood those babies who succumbed were kept alive in the order reported in Table 1, for twelve, eight, eleven, fourteen and thirty days. In view of the prematurity and other handicaps suffered by these babies at the onset of their illness they were rapidly exhausted by the constant vomiting and diarrhea. We were aware that the supportive measures of glucose, fluid and blood administration were only partly meeting the demands of this disease. An attempt was made to supply vitamins in concentrated form, but it is doubtful whether much if any was absorbed. As their general resistance was lowered, one after another acquired some sort of pyogenic infection. Two infants (Cases 1 and 6) developed subcutaneous infections at the sites of repeated injections. Nearly all the infants acquired some kind of respiratory infection. It is interesting that there were relatively few stools in comparison with the toxemia and prostration. An extremely trying feature in the clinical course was the severe and persistent abdominal distention. The cause appeared to be an adynamic condition of the intestinal musculature rather than excessive fermentation.

TABLE 1. *Summary of Clinical Courses.*

CASE NO.	STOOLS	THERAPY	FEEDINGS	SYMPTOMS AND COMPLICATIONS	OUTCOME
1	8–10 daily; yellow, soft to yellow-green, watery.	Clyses and intravenous glucose; prostigmin.	Pectin-agar; protein milk; starvation with rice water.	Temp. 100–102°F.; vomiting; distention; convulsions; cellulitis; terminal pneumonia.	Death on 12th day; weight 5 lb., 4 oz.; autopsy refused.
2	4–6 daily; yellow, soft to green, watery.	Clyses; subcutaneous blood; prostigmin.	Dilute evaporated milk; raw scraped apple; protein milk.	Temp. 99–100°F.; distention; vomiting; probably terminal pneumonia.	Death on 8th day; weight 4 lb.; autopsy refused.
3	6–8 daily; yellow, loose to green, watery.	Clyses; subcutaneous blood; prostigmin; buffer solution.	Olac; dilute pectin-agar; rice water.	Temp. 102–103°F.; vomiting; distention; hematemesis; terminal pneumonia.	Death on 11th day; weight 4 lb., 11 oz.; autopsy obtained.
4	6–8 daily; yellow, loose to green, watery.	Clyses and intravenous glucose; transfusion (60 cc. citrated blood).	Olac; pectin-agar; rice water; protein milk.	Temp. 102–104°F.; vomiting; distention; terminal respiratory complications.	Death on 14th day; weight 4 lb., 5 oz.; autopsy obtained.
5	5–9 daily; yellow, loose to green, watery.	Clyses; transfusion (60–100 cc. citrated blood); starvation 48 and 60 hr.	Stepped-up protein milk; rice water, dilute skimmed milk.	Temp. 97–103°F.; distention.	Complete recovery; discharged on 25th day; weight 6 lb.
6	4–8 daily; yellow, loose to green, watery (formed before death).	Clyses and intravenous glucose; 4 transfusions (40–100 cc. citrated blood); starvation 48 and 72 hr.	Dilute skimmed milk; breast milk; rice water; stepped-up protein milk.	Temp. 99–103°F.; vomiting; distention; cellulitis; probable pneumonia.	Death on 30th day; weight 4 lb., 13 oz.; autopsy obtained.

The following pathological summary is based on autopsies performed by the pathologist, Dr. James Beck, on Cases 3, 4 and 6. The findings in the gastrointestinal tract were remarkably similar in each case and closely resembled those reported in other series. They were notable for their mildness. The mucosa of the stomach appeared pale and was covered by a thin film of mucous material. The duodenum showed a moderate injection. The mucosa of the entire intestinal tract was thin, yellowish and smooth. There were no ulcerations or areas of hemorrhage. The loops of the bowel were distended by an odorless gas. Peyer's patches showed no enlargement. The mucosa of the colon was negative. Microscopically there was a minimum change throughout. This consisted of a moderate edema of the entire wall of the bowel, with only slight and scattered areas of leukocytic infiltration. There appeared a slight distention of the intestinal vessels, with diapedesis of blood corpuscles and hemorrhage in places. In Case 6, in which peritonitis was found, the serosa showed a mild inflammatory reaction and was covered with a small amount of exudate. All 3 of the autopsied babies showed parenchymatous degeneration of the liver, kidneys and heart, with fatty degeneration of the liver. Cases 3 and 4 showed edema of the lungs, with aspiration bronchitis from foreign material. Besides peritonitis, Case 6 showed a bilateral empyema and the congenital anomalies of cleft palate and left harelip, right cryptorchidism and Meckel's diverticulum.

The finding of *Bacillus welchii* as the predominating organism in the stools of several babies in our series is not regarded by us as being significant. No clinical or bacteriological evidence

of the amebas or bacteria commonly found in diarrhea could be discovered in these cases. In view of the highly infectious nature of this outbreak and the extremely mild pathologic findings, in striking contrast with the severe toxicity of the infection, we strongly favor a virus infection as the primary cause. Studies made of the throats of contacts of the babies and of their foods and various fomites were unproductive in revealing the cause or method of spread of the infection.

Accepting the contention that infectious diarrhea of the newborn, as reported by Rice and others, is a disease entity, we have every reason to believe that our cases were of the same nature. Both the clinical course and pathologic findings support this view. Moreover Case 1, which was admitted to the Pediatric Service with a diarrhea already well advanced and was undoubtedly the source of infection for the other infants, had been discharged only a few days previously from a New York hospital. The superintendent of this hospital stated that before the infant's discharge she was in a ward of the hospital in which there was a case of infectious diarrhea. Knowing the highly contagious nature of this infection, we are inclined to believe that our cases originated in this manner. It is interesting to note that since this outbreak we have had no similar cases in our hospital, nor have other cases been seen by me or any of my colleagues in this area. We believe that this is the first instance of this infection to be reported in New England. However, we have heard unofficially of one or two possibly similar outbreaks. We think it important to call attention to the highly contagious nature of this disease, as a warning should other outbreaks occur.

Although our therapeutic efforts were unsuccessful in all but one case, our negative observations may perhaps be of value. Periods of partial starvation with fluid administration by mouth in the form of tap water, cereal water or saccharin-sweetened tea were entirely unsuccessful in controlling either diarrhea or vomiting. Various feedings such as dilute evaporated or skimmed-milk mixtures, protein milk, a commercial pectin-agar preparation and raw scraped apple were all ineffectual in checking either diarrhea or vomiting. Reliance had to be placed on parenteral feedings to keep the babies alive. An approach advocated by members of the house staff was accepted by the visiting staff only after other measures had failed: to submit them to periods of complete starvation lasting from forty-eight to seventy-two hours. In view of the infants' condition this seemed a decidedly hazardous procedure, but in every case the diarrhea was checked. A recurrence demanded another period of starvation. This program was undoubtedly responsible for the recovery of Case 5. The stools in Case 6 became formed after two periods of starvation and remained formed up to the time of death, which was caused by pyogenic complications. This procedure is safe only when extremely vigorous measures of fluid administration are carried out. We came to the definite conclusion that an earlier use of this measure might have saved the lives of more of the babies. Following periods of starvation the use of protein milk in minute and increasing doses appeared to be the most successful method of feeding. In treating the severe abdominal distention relatively large doses of prostigmin proved of most aid.

SUMMARY

A disastrous outbreak of diarrhea on the Pediatric Service of the Worcester Memorial Hospital is reviewed. Nothing positive is known of the infecting organism and therefore of the method of the spread of the infection. Ordinary ward procedures, however, proved ineffectual in controlling the infection.

The need is emphasized of regarding diarrhea occurring in this susceptible age group with the utmost concern. The safety of other contacts demands that such cases be isolated, under the most careful aseptic, individual nursing care.

The clinical aspects of the outbreak are reviewed, and a picture of the relatively mild pathologic findings is given.

The value of periods of absolute starvation followed by gradually increased feedings of protein milk is emphasized.

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REPORT ON MEDICAL PROGRESS

GENERAL ANESTHESIA*

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A SUFFICIENT period has now elapsed since the introduction, some five to ten years ago, of certain anesthetic agents for some crystallization of thought concerning their safety and usefulness. While in some cases a fair consensus has been arrived at, in others opinion is still sharply divided.

VINYL ETHER

When vinyl ether was introduced some six years ago considerable difficulty in its use was experienced by many men. During induction there was considerable mucus and there were tremors and even convulsions. There was uncertainty about the drug's effect on the liver, owing to the demonstration of liver injury in experimental work on dogs under prolonged anesthesia. These various difficulties, however, are now rarely experienced. Possibly some of this change is due to the greater purity of the drug now produced. Goldschmidt, Ravdin and Lucké,[5] however, have shown that liver injury does not take place in the presence of abundant oxygen. This is simply another example of the damaging effect of anoxemia on the liver when toxic agents are being used and of the protective action of abundant oxygen, an action already frequently stressed by various investigators, but still occasionally ignored by many clinicians.

While general opinion is still somewhat divided on the use of this drug, it has on the whole had a favorable reception.[1, 6, 9] The facts that it is only very slightly irritating to inhale, that anesthesia may be induced with great rapidity, and that recovery is rapid and generally favorable, together with its small bulk, easy portability and the lack of necessity for bulky apparatus or equipment, make it very useful in a number of circumstances. It may readily be given with a simple open cone, and makes a pleasant, rapid and smooth induction for ordinary ethyl ether. It is applicable for short general anesthesia when relaxation is desired and for these operations and for ether induction is preferable to ethyl chloride because of its greater safety. While it lacks the dangerous vascular action of ethyl chloride, however, it resembles the latter in being very rapid and powerful in its action, and consequently it should be used with great care.

BARBITURATES

Opinion concerning the safety and use of the intravenous barbiturates is still divided, but they are being extensively used, and their employment seems to be increasing rather than diminishing. The increasing use in the operating room of electrical apparatus such as diathermy machines, which form potential ignition for inflammable anesthetics, together with some explosions of the latter, has focussed attention on this aspect of anesthesia and has emphasized the value of anesthetic agents such as the barbiturates, which are entirely free from danger of fire or explosion.

It is pointed out by those who oppose the use of these drugs that they resemble hypnotics rather than true anesthetics, and that the margin between the anesthetic and the lethal doses is comparatively narrow. This fact would tend to limit their use to operations which are either quite short or require only a light plane of anesthesia. It is also pointed out that their elimination is not so rapid as is that of the inhalation anesthetics. This is quite true if by the latter is meant the gas anesthetics such as nitrous oxide and cyclopropane. The difference in recovery time between ethyl ether and the intravenous barbiturates, however, is not great. Since they depend on the liver and kidneys for their detoxification and elimination they should not be used when there is severe disturbance of the function of these organs. There is a reasonable question whether elimination would not be greatly slowed up if shock should supervene.

Lundy has repeatedly pointed out that their use in children is inadvisable, but Hudson[7] has reported favorably on the use of Evipal in a series of 100 cases in infants and children.

With lethal doses, death takes place from respiratory rather than cardiac failure. This emphasizes the necessity for an experienced anesthetist and proper equipment for efficient artificial respiration, and again demonstrates the value of abundant oxygen, as well as the necessity for a competently functioning liver. Immediate depres-

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sive effects are proportional to the rate of administration rather than to the total dose.

These drugs are being increasingly employed also as useful adjuncts to other forms of anesthesia. With local and regional anesthesia and with spinal anesthesia they are used by some for a quieting effect, sometimes without even the production of actual unconsciousness. Others employ them in combination with nitrous oxide to obtain a fireproof anesthesia of greater depth than seems advisable when either is used alone.

The two intravenous barbiturates most commonly used, the sodium salt of ethyl- (1-methylbutyl) thiobarbituric acid (Pentothal Sodium) and the sodium salt of cyclo-hexenyl-N-methylbarbituric acid (Evipal Soluble), while similar in many respects, are so different in many ways that in clinical use they may be regarded almost as complementary, rather than as interchangeable. Pentothal is powerful and depressing, and produces a smooth induction and good relaxation. Evipal is not so depressing, but induction is often marred by muscular tremor, and relaxation is apt to be poor. Pentothal is stated to produce liver necrosis in mice, while Evipal appears to be free from this hazard.[10] Further investigation of the action on the liver is now in progress, and while final results are not now available, it appears highly probable that when the liver is well supplied with glycogen and when there is no lack of oxygen during and following anesthesia, liver damage need not be feared. It is thus advisable to avoid any trace of anoxemia during the use of Pentothal as well as with other non-volatile general anesthetics.

The 10 per cent solution of these drugs which was at first recommended sometimes produced necrosis of tissue if accidentally deposited outside the vein. The 5 per cent solution now employed often causes a tender induration if spilled outside the vein, but does not ordinarily produce necrosis.

CYCLOPROPANE

Probably no other anesthetic has ever received such a widespread and enthusiastically favorable reception as has been that accorded this one. Since its introduction by Waters a voluminous literature has appeared which has given enthusiastic approval. Anesthetists everywhere have been most favorably impressed with its characteristics, such as very low toxicity and the large amount of oxygen which can be used with it. While comment is still very favorable it has become slightly tempered and is somewhat more discriminating. It is realized that no drug is perfect, and that this one is so powerful and depressing that untoward and even alarming results may follow if it is given too rapidly or to too great a depth. To prevent these occurrences some clinics have adopted certain rules, such as that the rate of flow of the gas shall never exceed 500 cc. per minute, or that the rate of cyclopropane flow shall never exceed that of oxygen.

Only one distinctly unfavorable report has appeared.[2] This records 4 cases of collapse under cyclopropane, with 2 deaths. One of these deaths, however, was largely due to other causes. The opinion is expressed that this present chapter of anesthetic history may be labeled "Cyclopropane: A surgical tragedy." This report, however, and this opinion stand alone. Nothing similar is recorded elsewhere.

At the Lahey Clinic we have had 3 deaths among 10,000 patients under cyclopropane anesthesia, 1 of them due to explosion.* In order to see what the experience of others has been and to test present informed opinion, I have canvassed a number of the large users of cyclopropane. The replies indicate that they have used this anesthetic in 30,000 cases and have had 5 deaths attributable to the anesthetic. This, together with our experience, gives 40,000 cases and 8 deaths. If our death due to an explosion is eliminated, the mortality rate is found to be 1:5700. One of these users, who had done 5000 cases under cyclopropane without a death, stated that there had been 5 deaths under other anesthetics during the period when the former cases were being done. All these men still favor the use of cyclopropane, some of them quite enthusiastically.

The death from an explosion of this anesthetic, which recently occurred, has focussed attention on this aspect of the gas, and a meeting was held in Boston to formulate a general policy in regard to its use. It was attended by experts on electricity and explosives from the Massachusetts Institute of Technology and by surgeons, anesthetists, hospital executives and trustees of Greater Boston. After a free and thorough discussion bearing on all aspects of the explosiveness of cyclopropane and other anesthetics, it was decided to take no action looking to any curtailment of its use.†

At present, then, it is safe to say that experience with cyclopropane over the last five years has shown it to be a powerful drug which should be employed with great care and only by those well versed in its use, but that its properties in other respects are so valuable that informed opinion is now strongly favorable to its continued and rather extensive use.

*The 2 occurring before the explosion were reported at the meeting of the Section of Anesthesia, Canadian Medical Association, Halifax, June 22, 1938.

†A summary of the discussion will be published shortly.

The intratracheal method with this and with other agents is rapidly coming into more extended use and is now commonly employed at all well-equipped clinics.

NITROUS OXIDE

This gas has been very widely held to be one of our safest general anesthetics, if not the safest. This view is to a very large extent justified because of the fact that the gas is of very low toxicity, and because it is eliminated with such extreme rapidity that resuscitation from overdosage is usually easy. However, it is such a weak anesthetic that if used without an adjuvant such as ether it has to be pushed very vigorously in order to obtain sufficient anesthesia. This cuts down the allowable oxygen to the point where there is but a very narrow margin between anesthesia and asphyxia, and considerable skill is therefore required to hold the patient within this range. While resuscitation is very rapid and effective if oxygen is given in time, collapse is equally rapid if it is not so given. Many anesthetists have felt that for this reason pure nitrous oxide and oxygen was not the superlatively safe anesthesia it was generally taken to be, and that it was frequently better to add some sort of adjuvant, usually ether, thus allowing more oxygen to be used.

Courville[3] was the first to crystallize thoughts that had been in the minds of many, by presenting postmortem proof of extensive brain injury in 13 fatal cases. His work has been confirmed by others,[8] and Courville[4] has now demonstrated that the cerebral lesions are due to asphyxia rather than to any toxic property of nitrous oxide itself. Stewart[11] again emphasizes these facts by reporting a case of cerebral necrosis and death following apnea during nitrous-oxide and oxygen anesthesia. "Neurologic symptoms," he writes, "appeared within an hour and lasted about forty-eight hours, when coma supervened. Death from pneumonia occurred on the thirteenth postoperative day. A review of the literature shows that this is not a rare sequence of events."

* * *

This report on progress in general anesthesia seems to resolve itself into a testimonial to the value of oxygen. Its action in protecting the liver has been shown with vinyl ether (as well as with chloroform and other toxic agents). It is of great value in anesthesia with the barbiturates (as well as with Avertin). It seems highly probable that the low clinical toxicity of cyclopropane is due in no small measure to the abundant oxygen ordinarily used with that drug. The harmful effects of lack of oxygen are shown with pure nitrous-oxide and oxygen anesthesia. Altogether, this emphasizes the fact that oxygen is a life-giving necessity, and that abundance of it in general terms means safety, and lack of it danger.

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CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

ANTEMORTEM AND POSTMORTEM RECORDS AS USED IN WEEKLY CLINICOPATHOLOGICAL EXERCISES

FOUNDED BY RICHARD C. CABOT, M.D.

TRACY B. MALLORY, M.D., *Editor*

CASE 25161

PRESENTATION OF CASE

Third Admission. A fifty-eight-year-old freight clerk entered the hospital for repair of a right inguinal hernia. In his family history it was noted that his mother had died of arthritis and cardiorenal disease. This was the third admission for the patient, but the records for the two previous ones had been lost. No information could be obtained regarding the first admission, except that as a child he had had a periostitis of the tibia for about three months. At the second admission a posterior gastroenterostomy for duodenal ulcer was done. There was complete relief of symptoms thereafter.

The other item of interest in the past was a story of "inflammatory rheumatism," since his early thirties. Because of this condition he had been forced to stay out of work at least two or three weeks a year. The feet were usually affected, but in recent years he had complained of arthritis of the knees and hands. He was never permanently crippled. Two weeks before this admission he began to have another attack of "rheumatism" in his feet and knees and as he was unable to get about, he thought it a "good time" to have his rupture repaired.

Physical examination showed bilateral inguinal hernias and stiffness of both knees, with some limitation of extension. The right foot was swollen, and over the dorsum of the first metatarsal there was some tenderness. The blood pressure was 120 systolic, 75 diastolic. There was a soft systolic murmur at the apex, although the heart was not enlarged to percussion.

Examination of the urine showed a specific gravity which varied between 1.008 and 1.028. There was a very slight trace of albumin on three examinations. No casts or red cells were seen in the centrifuged specimen. The nonprotein nitrogen was 32 mg. per 100 cc.

X-ray examination of the knee showed slight increase in the density of the bones with no change in the joint spaces. The elbow joints were normal.

A medical consultant noted that there was little in the way of deformity after twenty-five years of rheumatic attacks. It was suggested that a search for foci of infection be made. Both hernias were repaired, and the only complications to his hospital stay were herpes simplex and retention of urine. During the study of his urinary retention he admitted having passed on many occasions a urine which on settling showed a brick-dust deposit in the bottom of the vessel.

Fourth Admission (five months later). Following an accident he had a dislocation of the semilunar bone of the right wrist and a fracture of the styloid process of the right radius.

Fifth Admission (four years later). The attacks of acute arthritis continued as before. Three weeks before this entry he began to have pain and tenderness in his left foot and ankle, and his local physician advised him to enter the hospital so that pus might be drained from this area.

Physical examination showed a moderately inflamed area 3 cm. in diameter over the lateral surface of the fifth left metatarsal.

X-ray showed no evidence of osteomyelitis but some calcification of the blood vessels of the legs.

The tumor mass was removed surgically and on pathological examination showed chronic inflammation and uric acid deposits.

Sixth Admission (three years later). His "rheumatism" had progressed somewhat, and in addition to two or three acute attacks of arthritis each year, he had a great deal of stiffness of his joints all the time. Two weeks before admission he was forced to go to bed because of swelling of his right knee and dropsy of the right ankle.

Physical examination showed swelling and some tenderness of the knee, although there was no edema or joint effusion. There was some weakness of the right leg. The temperature, pulse and respirations were normal.

An orthopedic consultant examined the patient and expressed the opinion that the patient was suffering from rheumatoid arthritis.

Examination of the urine showed a specific gravity which did not exceed 1.014 at five examinations. A very slight trace of albumin was observed on one occasion. No casts or red cells were seen. A blood Hinton test was negative. The nonprotein nitrogen was 23 mg. per 100 cc. Three blood uric acid determinations were 1.7 mg., 5.5 mg. and 4.2 mg. per 100 cc. respectively. The basal metabolic rate was +13 per cent.

X-ray films of the spine showed no evidence of disease of the vertebral bodies but a localized process suggestive of trauma. There were proliferative changes about the knees and the ankles.

Seventh to Tenth Admissions (during the next

five years). During this time he was admitted three times to the hospital. On one occasion it was for a strangulated hernia, and the others for the treatment of his arthritis. At each admission it was felt that he was becoming progressively weaker and had more dyspnea on exertion. His activity was becoming progressively restricted. While he was being followed in the Out Patient Department for the distress in his feet, he was given foot pads and shoe supports, which appeared to make his feet worse and caused him to discard them.

The maximum specific gravity of his urine which was observed during this interval was 1.012. The urine contained some albumin but no casts and only rare red cells. Two years before his last admission x-ray films of the feet showed a marked narrowing of the joint spaces, with hypertrophic changes. No areas of destruction were seen. The knees showed changes which were thought to be characteristic of proliferative arthritis. X-ray films of the spine showed hypertrophic changes typical of the mixed form of arthritis.

One year before his last entry the patient spent the summer traveling to the West Coast and said that he had enjoyed the trips as much as he had when he had gone twenty years before.

Tenth Admission (one year later). He was admitted for a study of his kidney function and on this admission, inulin and creatinine clearance studies were done. An electrocardiogram taken at that time showed A-V and I-V block, indicative of widespread myocardial damage. Examination of his heart showed a rough systolic murmur heard all over the precordium. The blood pressure was 138 systolic, 78 diastolic.

Final Admission (two months later). He was admitted because of progressive shortness of breath and inability to care for himself at a friend's house where he had made his home. Examination showed no evidence of cardiac decompensation or pulmonary edema. The joints were essentially unchanged.

The electrocardiogram showed no change from the previous examination. The serum nonprotein nitrogen was 100 mg. per 100 cc., the serum uric acid 13.8 mg. The total fixed base was 142 milliequivalents; the sodium, 132 milliequivalents; the carbon-dioxide combining power, 23 vol. per cent; the phosphate, 5.1 mg. per 100 cc.

He became comatose shortly after admission. Two days later, without regaining consciousness, he developed hiccoughs and urea frost, and died following a terminal elevation of temperature to 104.8°F. and of pulse rate to 160 per minute.

Differential Diagnosis

Dr. Alfred Kranes: It seems to me there are two questions to be answered in this case: What form of arthritis did the patient have? What type of renal disease? Before we discuss the arthritis, I think it might be interesting to point out one or two factors in this history. In the first place, it is interesting to note that his mother had died of the same disease or what appeared to be the same disease as the patient — some kind of joint disease and nephritis. Another interesting feature is that this patient had rheumatism for about forty years, and during that period he was apparently never very much crippled by it. It is stated that despite the long history of joint disease he got along fairly well. Another interesting point in the history is that his arthritis probably came in attacks, although the history is not very clear on that point. I am not sure just how long the attacks lasted, despite the statement that he was out of work two or three weeks each year because of them. It would also be of interest to know what precipitated the attacks, if anything did, and in addition whether he was perfectly free from any joint symptoms between acute attacks.

So far as the laboratory work goes, I get very little help there. The x-ray films are said to have shown hypertrophic changes, but at his age one would expect them. After all, he died at the age of seventy-two, and most of the studies were carried on in the last ten or twelve years of his life.

I should like to see the x-rays.

Dr. Aubrey O. Hampton: These x-ray films are more interesting than the note would lead one to believe. I shall start on something I think I know a little about. The heart is enlarged, and the enlargement is left ventricular, with a tortuous aorta and calcification of the arch. There is also evidence of peripheral arteriosclerosis, which is seen even down to the terminal phalanges of the feet. When were these films taken?

Dr. Kranes: I believe in 1936. He was seventy years old then.

Dr. Hampton: The changes in the spine are those of the ankylosing type, with large spurs crossing the joints and with fairly normal joint spaces. This is not what one would expect with rheumatoid arthritis but rather the changes that go with the degenerative type. The lumbar spine shows practically nothing. The distribution of disease in the other joints is spotty. All the metatarsophalangeal and phalangeal joints are normal except for those of the first toes, while the tarsal joints are grossly abnormal. There are joint destruction and fusion in these tarsal joints, certainly

more marked lesions than degenerative arthritis would produce. The joint spaces are entirely absent, and it is an ankylosing type of arthritis. The great toes at the first metatarsophalangeal joints show punched-out areas of bone destruction that certainly simulate gout. There are smaller areas in the posterior joint margins of the ankle which go with gout. In this elbow there is calcification in soft tissue with swelling at one time, and calcification without swelling the next time. At no time is there as much atrophy in the bones as one would expect. From the x-ray appearance he did not have a persistent arthritis. He used his joints between attacks.

Dr. Kranes: Apparently there is a good deal more in the x-ray films than in the written report. I do not believe that we have to consider many forms of arthritis very seriously. In fact there is only one that need be considered at all, namely gout. I just cannot see how any other form of arthritis could give this history plus the x-ray findings. To be sure he probably did have a lot of degenerative arthritis, but you would expect it in any man of seventy-two, regardless of what other disease he might have. Rheumatoid arthritis is extremely unlikely with a history of this type and with these x-ray pictures. After forty years of rheumatoid arthritis, coming in attacks which were apparently quite severe, one would expect much more crippling or disability and a lot more in the way of x-ray changes in other joints. Other types of specific infectious arthritis, such as gonococcal, tuberculous and syphilitic, must be mentioned, merely to exclude. They are extremely unlikely, as is rheumatic fever. I do not think we have to consider anything aside from gout.

In favor of gout is the family history of joint disease which occurs in a very high percentage of patients with gout. The harder you look for it the more often you find it. The patient's history of acute attacks that subsided and left him relatively free between attacks is also quite characteristic. There is probably some increase of urate excretion in the urine, qualitative to be sure. The statement about brick-dust deposits in the urine probably represents precipitated urates, and patients with gout have larger urate excretions than do normal people. Finally there is the episode of an apparently septic joint. Acute gouty joints frequently resemble septic joints and are operated on with the idea that pus will be obtained. In this case uric acid deposits were found, which clinch the diagnosis.

There are nevertheless a few disturbing factors so far as the diagnosis of gout goes. First are the repeatedly normal uric acid determinations on his blood, except for the terminal one of 13.8 mg. per 100 cc., which, since the patient was in uremia, is of no diagnostic value. So far as I know, no cases of gout have been encountered in this hospital with a normal amount of uric acid in the serum. Other people working in this field disagree and claim that normal uric acid values occur in many gouty individuals. Dr. Talbott does not believe that and I would like to have him say a few words if this patient proves to have gout. In this connection it occurs to me that this patient had been studied over a long period of time and perhaps most of the uric acid determinations had been done by the old method. A second disturbing feature is that after forty years of gout he had developed no tophi. Thirdly, after two operations he developed no acute arthritis. Gouty attacks frequently occur after trauma and after an operation; however, this patient had two operations with no flare-up of his joint symptoms.

So far as his renal disease goes, I think it is always a guess to try to predict what type the patient has. In this patient, our first indication that he had any renal impairment occurred during his sixth admission at the age of sixty-five when it was found that his specific gravity did not go above 1.014. From that time until his death there was a slow progression of renal failure. The results of the clearance tests are not given, but I presume they were quite low, since he died two months later in uremia. I do not believe that this patient could have had glomerulonephritis at his age; at least there is no evidence of glomerular damage or of acute nephritis in the history. He was followed for a long period when his renal function was apparently normal. He was at the age of course when he might have had prostatic obstruction. However, there is no history suggesting it, and no mention on physical examination about the size of the prostate. People with gout frequently have uric acid stones. This patient had no history of having had attacks of renal colic. It is conceivable that he may have had silent stones blocking off one or both ureters and causing hydronephrosis. In the absence of direct evidence we have no right to make that diagnosis.

Dr. Hampton: He had a perfectly negative intravenous pyelogram in 1936. The kidney pelves were large, but the calices were not dilated and he functioned fairly well.

Dr. Kranes: That is interesting because according to the renal function tests done before 1936 he showed impairment of concentrating power.

How about a gouty nephritis? It is well known

that a very high percentage of people with gout develop renal insufficiency, but there is not so much agreement as to the type of renal lesion that occurs. Various authors describe different types. The most consistent lesion is a vascular type of nephritis. A patient of this age with long-standing gout and evidence of arteriosclerosis throughout his body undoubtedly has a high degree of vascular nephritis. It is queer that he did not have a higher blood pressure with a diffuse vascular nephritis. There is another type of renal lesion described in gout where deposits of urates occur in the renal tubules, causing a type of renal insufficiency similar, I suppose, to the Bence-Jones kidneys of myeloma; the kidneys becoming plugged with urates. There have been a few cases described in which these urate deposits have been primarily responsible for the renal failure. I wonder if this patient, with a normal blood pressure, did not have an extensive degree of this type of nephritis. He also had a severe degree of coronary arteriosclerosis, as shown by the electrocardiogram and by the enlargement of the heart without hypertension. So I shall summarize by saying that this patient had gouty arthritis, had probably a combination of vascular and gouty nephritis, whatever that is, had severe coronary disease and probably some terminal infection such as pneumonia; having died in uremia there is a strong possibility that he may have had terminal pericarditis.

Dr. John Talbott: Dr. Kranes is perfectly right in assuming that the earlier uric acids were done on whole blood. It is not unusual to find normal whole-blood uric acid, which if repeated on serum is elevated. There was one determination on whole blood (5.2 mg.) which is above the normal range. The level for serum uric acid of patients with gout is 6.0 mg. per 100 cc. or more.

So far as the x-ray films are concerned, in many cases we have given up trying to confirm a diagnosis of acute gouty arthritis by x-ray examination. I think this is a good example. The most we can say is that the changes are rather consistent with gouty arthritis and the fact that he had had his symptoms some forty years and did not have a characteristic picture does not mitigate against the diagnosis. The clinical appearance of his hands was that of advanced hypertrophic arthritis, as opposed to gouty arthritis with subcutaneous tophi that are characteristic. He had two tophi on his feet, however, which were removed surgically. The pathological report showed urate crystals. His kidney function by inulin clearance was only one-third normal. We believed that the course of the changes in the kidneys was consistent with gout. He had evidence of renal insufficiency in one form or another, without developing terminal failure except the last few days.

Dr. Hampton: How often do you see ankylosis in gout?

Dr. Talbott: In about 20 per cent of our patients.

Dr. Hampton: That is the thing that disturbed the X-ray Department. I do not remember ever having seen complete ankylosis of multiple joints as seen here in both tarsal areas. It upset me no end.

Dr. Talbott: We have several patients that show it.

Clinical Diagnoses

Gout.
Coronary heart disease.

Dr. Kranes's Diagnoses

Gout.
Vascular and gouty nephritis.
Generalized arteriosclerosis.
Terminal infection (? pneumonia, ? pericarditis).

Anatomical Diagnoses

Gout.
Hypertrophic (degenerative) arthritis of the spine.
Urate deposits in kidney.
Healed pyelonephritis, left kidney.
Chronic vascular nephritis.
Amyloidosis of kidneys and adrenal glands.
Cardiac hypertrophy.
Arteriosclerosis, coronary, aortic and renal.
Lobar pneumonia, right lower lobe.

Pathological Discussion

Dr. Tracy B. Mallory: At the postmortem examination all the joints examined, and they were many, showed extensive urate deposits—an extremely characteristic picture of gout. There was also hypertrophic arthritis of the spine. The remaining findings were a terminal pneumonia, a considerably hypertrophied heart, fibrotic and calcified but not particularly narrowed coronary arteries, and a pair of kidneys of which the left one weighed only 75 gm. and the right 275 gm. The left kidney was very characteristic of a healed pyelonephritis. The other kidney showed a combination of lesions. There were extensive deposits of urates in the pyramids. There were fairly marked blood-vessel changes. There was also a very considerable amyloid deposit. Histologically the adrenal glands also showed moderate amyloid disease. To which of all the multiple factors the renal insufficiency was due, I do not know, prob-

ably to a combination of all three. One might speculate that the adrenal involvement had prevented the development of hypertension, in view of recent work on adrenalectomy in Goldblatt dogs.

CASE 25162

Presentation of Case

A thirty-seven-year-old married woman was admitted complaining of swelling of the abdomen.

Two and a half years before admission the patient had been delivered normally of her fourth baby. Following this she had sacroiliac pain. A corset and a reducing diet were prescribed, but about three or four months later she began to eat freely again. During the next two years she gained weight progressively but noted no essential abnormality until two months before admission. During the two months preceding entry her abdomen became unusually large and was thought to be abnormally hard, particularly on the left side. A mass seemed to occupy the entire lower abdomen and extended into the left upper quadrant. She noticed a sense of pressure within the abdomen, but there was no pain except for a transient stinging sensation just below the left costal margin. Her menstrual cycle had remained normal, and there were no urinary complaints. There had been slight gaseous distention and increasing fatigability. Bowel movements were normal.

Physical examination showed a fairly obese woman with slight pallor. Examination of the head and chest was negative. The blood pressure was 125 systolic, 85 diastolic. Occupying the entire left side of the abdomen was a large, rounded, non-tender, questionably cystic tumor, which was not movable. It was thought that a lower border could be palpated above the brim of the pelvis. It was seemingly, though not definitely, continuous with a similar mass in the right lower quadrant. No further details were recorded. By vaginal examination the mass could not be palpated. The cervix was slightly eroded but in normal position, and the uterus was normally mobile.

The temperature was 99°F., the pulse 60, and the respirations 20.

The urine examination showed a specific gravity of 1.020, a large trace of albumin and a rare white cell. A phenolsulfonephthalein kidney-function test showed 50 per cent excretion in two hours. The blood showed a red-cell count of 4,570,000 with 80 per cent hemoglobin, and a white-cell count of 7300 with 60 per cent polymorphonuclears. The nonprotein nitrogen of the serum was 23 mg. per 100 cc. A blood Hinton test was negative.

A plain x-ray film of the abdomen showed a large, dense, smooth, rounded mass filling the entire left side of the abdomen and extending anteriorly across the midline in the lower abdomen. There were two 1.5-cm. flecky areas of calcified density in the right upper quadrant, but no areas of calcification in the mass. All the intestinal shadows lay in the right upper abdomen. An intravenous pyelogram showed a normal right kidney. The films did not show sufficient penetration to visualize the left kidney. There was a pressure defect on the fundic portion of the bladder, which was more marked on the right side. A barium enema showed no evidence of abnormality within the colon. The sigmoid was displaced to the right of the midline, and at the level of the sacroiliac joint there was a pressure defect suggestive of external pressure. The descending colon was displaced anteriorly and medially. On these films the left kidney shadow was sharply outlined and was not displaced; the renal pelvis and ureter were grossly dilated, and the ureter appeared to be compressed at the level of the second lumbar vertebra.

On the fifth hospital day an operation was performed.

Differential Diagnosis

Dr. Franklin G. Balch, Jr.: In going over the history, I cannot get much enlightenment as to the diagnosis. We have a thirty-seven-year-old woman who had gained considerable weight, particularly in the last two months. Physical examination showed that she was fairly obese and well-nourished and that there was a large mass on the left side, as described. The laboratory findings do not contribute much. She had a large trace of albumin in the urine which can be explained by damage to the left kidney. The x-ray films seem to be the most important things in reaching a diagnosis. I wonder if we can see them now.

Dr. George W. Holmes: One notices first a mass in the flank which is displacing the bowel toward the right. Apparently it is not connected with the gastrointestinal tract. I should suspect that the question was to decide whether it was retroperitoneal or in the peritoneal cavity.

Dr. Balch: Can you make out any definite borders?

Dr. Holmes: No.

Dr. Balch: Is that some of the dye retained from the previous intravenous pyelogram?

Dr. Holmes: This film must have been taken

on the same day that the dye was given and rules out the kidney as the source of the tumor. The kidney is displaced somewhat toward the spine and upward. The tumor would be retroperitoneal if my interpretation is correct. I am not quite certain how much the kidney is displaced because the film was taken with the patient lying face down. I think we can say that the tumor is not in the gastrointestinal or the urinary tract and that in all probability it is retroperitoneal. There is nothing in the chest.

Dr. Balch: That helps a good deal in the diagnosis. When I first read over this case, I thought of the possibility of ovarian cyst, but on further consideration, it did not seem to fit in with ovarian cyst because, as Dr. Holmes has pointed out, the colon is displaced medially. I do not believe that even a large ovarian cyst could do that. Also, this tumor could not be felt on vaginal examination, and we should expect to be able to palpate an ovarian cyst by this method. Fibroids are ruled out by the physical examination. Another possibility of intraperitoneal tumor is mesenteric cyst, which is a rare finding and is usually freely movable; therefore we can rule that out. Splenomegaly can be ruled out by the x-ray films and the lack of other findings. So that leaves us with retroperitoneal tumors to consider.

Malignant kidney tumors, such as hypernephroma and carcinoma, I believe we can rule out by the x-ray findings. Polycystic kidneys are usually bilateral, and the x-ray films do not suggest these lesions. A diagnosis of a single solitary cyst of the kidney does not fit. Extensive hydronephrosis is ruled out by the x-rays, even though there is evidence of a hydronephrosis. Retroperitoneal and intraperitoneal echinococcus cysts have been reported, but there is nothing here that would make us consider such a diagnosis. Therefore, we are left, it seems to me, with the diagnosis of a retroperitoneal sarcoma or, more likely, a retroperitoneal lipoma. With a retroperitoneal sarcoma of this size the patient should show a good deal more emaciation than she did and an advanced state of cachexia. It seems to me that limits our diagnosis pretty much to retroperitoneal lipoma. These are known to be slowly growing and frequently asymmetrical. They have been reported as weighing as much as 60 or 70 pounds, and usually start in the perirenal fat.

Dr. Holmes: May I interrupt to say that the x-ray picture is not one of lipoma.

Dr. Balch: That disturbs me. However, I still believe that it is a retroperitoneal tumor and not an intra-abdominal tumor. In spite of the x-ray findings I cannot figure out any other retroperitoneal tumor that would seem to fit this picture, so I shall stick to my diagnosis of retroperitoneal lipoma.

Dr. Tracy B. Mallory: Would anyone else like to hazard a diagnosis?

Dr. Augustus S. Rose: Was it definite that there was a second tumor in the right lower quadrant?

Dr. Mallory: That was not confirmed later.

Dr. George A. Leland: In addition to the x-ray report that was available when this patient was on the service last summer, I had the advantage of Dr. George G. Smith's examination, which is not recorded for Dr. Balch. I think what he said is of interest. The note is as follows: "The hydronephrosis on the left is probably due to pressure on the ureter by the cyst. This suggests retroperitoneal origin for the cyst. The right kidney seems normal. I do not believe there is anything about the kidney condition to indicate removal of the cyst."

In other words, those who had an opportunity to examine the patient were quite certain that there was a cyst. The protocol said that there was a questionable cystic tumor; that was misleading. The members of the X-ray Department likewise ventured into the realm of possibilities; they mentioned in their original report that "the difference in densities suggests lipoma or cyst." On account of the density of the mass, the possibility of sebaceous material was also mentioned somewhere in the history. So preoperatively we committed ourselves to a diagnosis of retroperitoneal cyst of the dermoid type. We thought that there might possibly be a developmental rest somewhere in this region.

I shall read my operative note:

> A 10- to 12-cm. incision was made through the left rectus muscle, the major portion of the incision being above the level of the umbilicus. Exploration with the examining hand disclosed a large lobulated cyst which was retroperitoneal. It had displaced the descending colon toward the midline. The uterus could be identified. The right ovary was normal in size and shape; the left ovary was elongated. The liver appeared to be normal. There was nothing abnormal in the peritoneal cavity to suggest malignant disease. A puncture wound was made in the cyst, and approximately 5500 cc. of clear serous fluid was aspirated with the suction apparatus. After this had been removed the anesthetist reported that the patient's pulse had gone up very rapidly; the suction apparatus was therefore removed. The opening was closed with interrupted catgut sutures. The peritoneum overlying the cyst was then divided, and the cyst was freed, chiefly by manual blunt dissection, from the descending colon and the region of the kidney. The kidney was visualized. Gradually the entire cyst was brought out through the opening and freed of its attachments. Altogether perhaps six or seven veins, none of which were over 2 mm. in diameter, were clamped. There was no main blood supply. After the cyst had been

delivered it was apparent it had no connection whatsoever with the ovary or parovarian structures. The peritoneum of the posterior wall was then brought together, and the wound was closed in layers, in the usual fashion. Evacuation of the contents of the cyst remaining after the cyst had been removed yielded about 2800 cc. Since a certain loss of fluid was inevitable when the stab wound was made, the cyst must have contained very close to 8000 cc. of fluid. On opening the cyst in the operating room there were two areas which resembled white coral; these areas were not hard.

The patient's convalescence was quite satisfactory.

Clinical Diagnosis

Retroperitoneal dermoid cyst.

Dr. Balch's Diagnosis

Retroperitoneal lipoma.

Anatomical Diagnosis

Retroperitoneal papillary adenocystoma.

Pathological Discussion

Dr. Mallory: The specimen that reached us was indistinguishable from an ordinary papillary cystoma of the ovary. It was lined with an epithelial layer, and there were short, densely fibrous papillary projections into the cyst. If I had not had Dr. Leland's word that the left ovary was found, I would have assumed, without question, it was an ovarian cyst. I do not know what its origin may have been unless some remnant of ovarian tissue had been left there in the course of the descent of the ovary. I cannot remember ever having seen this type of cyst in retroperitoneal tissue, in fact anywhere except in the ovary.

The patient had an interesting aftermath that might be brought up. A few months after operation a tumor of about half the size of the original cyst developed in the same location; this was very suggestive of a recurrence and caused a great deal of diagnostic difficulty. Dr. Holmes, have you anything to say on that?

Dr. Holmes: The mass is about the same density as that of the previous tumor. From the x-rays one is not justified in saying just what the character of the mass is. All we can say is that there is a small mass which has recurred in the same region.

I should like to point out that if it had been a fatty tumor it would have been of about the same density as air in the abdomen. Dr. Leland's statement was interesting; we are apparently growing up.

Dr. Mallory: After numerous examinations the patient was re-admitted and re-explored, and a cystic mass again found in approximately the same area. A section through the wall showed a hematoma with organization, so that it was not a recurrence.

Dr. Balch: The record stated that the large mass of the original tumor was continuous with a small mass on the right. Did you find any such mass?

Dr. Leland: As I recall it there was a sort of lobulation that went across the midline.

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CHIROPRACTIC AGAIN

The report of the Special Commission on Osteopathy, Chiropractic, Food, Drugs and Poisons has been filed as House Bill 2151. Among other objects, the "commission was created for the purpose of making an investigation relative to establishing a board of examination and registration in osteopathy, and regulating the practice of osteopathy; [and] relative to establishing a board of registration of chiropractors, and regulating the practice of chiropractic." The commission recommends that in the case of osteopathy no legislation is necessary, but that in the case of chiropractic a board of registration should be established, "but with very strict regulations." Three matters deserve discussion: the disposition of the problem presented by the petitions of osteopathic physicians; the reason why osteopathy and chiropractic did not receive the same treatment by the commission; and the "very strict regulations" for chiropractic set forth in the bill.

Concerning osteopathy, the report reads: "After several hearings the commission is not convinced that the Commonwealth should establish a separate board of examination and registration of osteopaths. It has been asserted, and admitted, that osteopaths engage in the practice of medicine; therefore it appears to the commission that they should be required to pass the examination of the Board of Registration in Medicine." The premises are sound; in logic, there is no escape from the conclusion.

Why does not the same argument apply to chiropractic? It has been asserted, and admitted, that chiropractors engage in the practice of medicine. Furthermore, the report reads: "Evidence was presented to the commission that there are many chiropractors illegally practicing in Massachusetts today, many of them being incompetent. For this reason we feel that a board of registration should be established. . . ." "Practicing" would seem to constitute the practice of medicine — an opinion upheld by the Supreme Judicial Court of Massachusetts in the Zimmerman case. If this be so, why should there be any discrimination between the statutory regulation of osteopathic practice and that of the chiropractors? Is it a good policy to legislate according to the dictum that the more illegal and incompetent practitioners there are, the greater is the need for lower standards of practice?

Restriction of space prevents discussion of all the details of the bill, but a critical point is found in Section 87F, defining chiropractic: "Chiropractic, or the system, method or science commonly known as chiropractic, or the practice of chiropractic, is hereby defined to be the science of palpating and adjusting the articulations of the human spinal column. This definition is inclusive, and any and all other methods are hereby declared to be not chiropractic." This is jargon triumphant. "Practice" or doing is defined as "science" or knowing. Furthermore, "the very strict regulations" appear to be unenforceable.

In spite of the strong arguments in the minority report on chiropractic, it remains the report of a minority, and as such, carries less weight, although the commission was unanimous against a separate board for osteopathy. The lesson to be learned from this report is that physicians must ever be alert to detect efforts to lower the standards for the practice of medicine and must constantly exert themselves to prevent such degradation.

ILLUSION AND SCIENCE

Modern science is characterized by a quality shown in William Harvey's investigations which led to his discovery of the circulation of the blood. He asked a critical question, *How much* blood is thrown out of the heart at each contraction? Today science asks insistently these questions: How many? How much? Mathematics, although it cannot be comprehended within the designation of the science of quantity, has served as an effective model for the approximately precise sciences of inanimate nature. It presents an ideal which the sciences of animate nature strive to realize. As some of the words or formulas are transferred to the social sciences, there is created an illusion of attainable exactitude and precision, dangerous because it is so far from actual knowledge.

A recent paper by Mr. Michael M. Davis,* entitled "Nursing Service Measured by Social Needs," is an example of this illusion, widespread in many social studies. The limitations of the value of statistics are well known to the expert and it is the disregard of these limitations which has led to the characterization of statistics as untruth of superlative degree. *Corruptio optimi pessimum.*

The purpose of the study was to obtain exact information concerning nursing service and methods of determining its quantitative and qualitative adequacy. This is indeed a social need. The writer uses the expression "yardsticks of both quantity and quality," and an example of the reasoning which leads to the "therefore" of his conclusions is given by the following quotation, in which the words in brackets are our own. It is a piece of reasoning invalidated by a premise which is admitted early and then treated as if it did not exist. "If we make an estimate of the amount of nursing care actually furnished, we do not find a reliable body of data [in logic the argument must stop if no reliable data are available, but not so in social science], but roughly from the known amount of unemployment of nurses in private duty and the amount of work done by salaried visiting nurses, it may be [unreliably] estimated that about one-fifth day's nursing care per capita of our population is now supplied the people of the United States outside of hospitals. If this figure is reliable [which it is not, *ex hypothese*], the amount of nursing now supplied outside of hospitals is about 40 per cent of the amount needed. If we had for each state and for many local communities such [unreliable] yardsticks of public need as compared with services rendered, we should be much farther along in judging what should be done, and where and how."

It is unfortunate that a plea for more science should be supported by unscientific argument. This kind of reasoning is indeed the strongest plea, unconscious perhaps on the part of the writer, for release from illusion and a return to the sanity which the word yardstick denotes but does not make real. Whatever other social needs there may be, one fundamental urgent need is restraint within the confines of logical discourse in dealing with our vital problems.

*Davis, M. M.: Nursing service measured by social needs. Am. J. Nursing 39:35-40, 1939.

MASSACHUSETTS MEDICAL SOCIETY

SECTION OF OBSTETRICS AND GYNECOLOGY*

Raymond S. Titus, M.D., *Secretary*
330 Dartmouth Street
Boston

Postpartum Hemorrhage

Mrs. H., a twenty-seven-year-old gravida III, at term, was seen at home in labor on the evening of September 10, 1922.

The family history was noncontributory. The pa-

*A series of selected case histories by members of the section will be published weekly. Comments and questions by subscribers are solicited and will be discussed by members of the section.

tient had undergone an appendectomy at twelve years of age and had had measles and mumps when she was twenty-three. Catamenia began at eleven, were regular with a twenty-eight-day cycle and lasted six days. She usually flowed profusely and with a great deal of pain on the first day. Her last period was November 26, 1921, making her due for delivery September 2. Her previous pregnancies had resulted in a normal delivery at term and a miscarriage at six weeks. The present pregnancy had been normal throughout.

When seen at home, the uterus was the size of a full-term uterus, the fetal heart was distinctly heard, and the vertex was presenting in the OLA position. The uterus was contracting every five or six minutes and relaxing well between pains. Labor had begun indefinitely at 5 p. m. A vaginal examination at 1 a. m. on September 11, showed the cervix more than half dilated, with intact membranes. The membranes were ruptured artificially, and the progress of labor became rapid. The baby was born shortly after 2 a. m. There was a median tear of the perineum at the site of a previous laceration. As soon as the baby was born, there was a large amount of fresh bleeding which showed that the placenta had separated. With each contraction, blood gushed from the vagina. Because of this, the placenta was immediately delivered by the Credé method. It came away intact, but the membranes were not complete. There was so much bleeding at the time that the perineum was not sewed up. Posterior pituitary extract, ergot and morphine were immediately administered, and a firm uterine contraction occurred; however, the patient went into profound shock. She perspired freely, was very white and asked for water. Her blood pressure was 80 systolic, 60 diastolic. She was given a quart of water by rectum, which was not retained. Heaters and blankets were applied, and the foot of the bed was elevated. There was no further bleeding. Her condition gradually improved, and at 7:30 a. m. she was in good condition, with a pulse of 100 and with much improved color. Her subsequent convalescence was uneventful save for a mild sapremia due to retained membranes.

Comment. This case is recorded because of the difficulties attendant upon home deliveries twenty years ago. The bleeding undoubtedly occurred because the placenta separated immediately and was not spontanously delivered. Even today such cases of profuse hemorrhage can hardly have the benefit of transfusion or the advantage of intravenous medication. Had this patient continued to bleed, the uterus undoubtedly would have been packed. Fortunately, added hemorrhage was not encountered; the uterus shut down adequately, and like most cases of shock alone, recovery was spontaneous.

Those of us who now do all our obstetrics in the hospital where modern facilities are available and where there are always plenty of hands to help cannot appreciate the difficulties and embarrassments of complicated home deliveries unless we are old enough to remember them. Undoubtedly, there are patients each year in Massachusetts who, confined at home, die of postpartum hemorrhage simply because the facilities of the modern hospital are not available. Some of the complications of obstetrics can be prepared for beforehand. Postpartum hemorrhage from uterine atony cannot be anticipated, and it is certainly not at all feasible to have the home equipped to meet this emergency; in consequence, hospitalization should be entertained whenever possible.

LEGISLATIVE NOTE

The chiropractic bill, now before the Committee on Public Health, has been given a number, House Bill 2151.

The people who favor this bill have been particularly active this year, and we strongly urge each member of the Society to write to his senator, his representative and members of the Committee on Public Health asking them to oppose it. The hearing will be held at 10:30 a. m., April 25, in the Gardner Auditorium. BE SURE TO COME, AND BRING YOUR FRIENDS!

CHARLES C. LUND, *Chairman.*
Committee on State and
National Legislation.

MEDICAL POSTGRADUATE EXTENSION COURSES

The following sessions, given by the Massachusetts Medical Society in co-operation with the Massachusetts Department of Public Health, the United States Public Health Service and the Federal Children's Bureau, have been arranged for the week beginning April 24:

BERKSHIRE

Thursday, April 27, at 4:30 p. m., at the House of Mercy Hospital, Pittsfield. Subject — The Control and Treatment of Respiratory Infections. (This is to include the serological treatment of pneumonia in infants and children.) Instructor: John A. V. Davies. Melvin H. Walker, Jr., *Chairman.*

FRANKLIN

Wednesday, April 26, at 8:00 p. m., at the Franklin County Public Hospital, Greenfield. Subject — Gonorrhea: Modern treatment of gonorrhea. Instructor: Sylvester B. Kelley. Halbert G. Stetson, *Chairman.*

MIDDLESEX SOUTH

Tuesday, April 25, at 4:30 p. m., at the Cambridge Hospital, 330 Mt. Auburn Street, Cambridge. Subject — Syphilis: Latent syphilis — diagnosis and treatment. Instructor: C. Guy Lane. Alexander A. Levi, *Chairman.*

SUFFOLK

Thursday, April 27, at 4:30 p. m., in John Ware Hall, Boston Medical Library, 8 Fenway, Boston. Subject — Anemia: Modern methods, diagnosis and treatment. Instructor: William P. Murphy. Reginald Fitz, *Chairman.*

APPLICANTS FOR FELLOWSHIP

PUBLISHED IN ACCORDANCE WITH THE PROVISIONS OF THE BY-LAWS (CHAPTER I, SECTION 1) AS AMENDED JUNE 2, 1938

BARNSTABLE DISTRICT

D'ELIA, ARTHUR J., South Chatham.
Tufts College Medical School, 1933.

NILES, JOHN O., Third Avenue, Osterville.
Harvard Medical School, 1936.

RICE, JACKSON M., Hyannis.
Middlesex College of Medicine and Surgery, 1932.

Donald E. Higgins, *Secretary.*

BERKSHIRE DISTRICT

ALLEN, PLINY A., 83 Chestnut Street, North Adams.
Harvard Medical School, 1934.

CHESANOW, ALBERT, 133 Castle Street, Great Barrington.
Middlesex College of Medicine and Surgery, 1928.

HAGOPIAN, LEON G., 1171st Co., CCC, North Adams.
Tufts College Medical School, 1911.

HOWARD, JAMES H., St. Luke's Hospital, Pittsfield.
University of Vermont College of Medicine, 1936.

NESBIT, CLAYTON W., 79 Lenox Avenue, Pittsfield.
Harvard Medical School, 1936.

WYMAN, EDWARD R., 244 Main Street, Great Barrington.
Tufts College Medical School, 1937.

Hugh J. Downey, *Secretary.*

BRISTOL NORTH DISTRICT

GILLIS, GRACE E., State Hospital, Taunton.
Tufts College Medical School, 1937.

PRINDLE, CLAIR G., State Hospital, Taunton.
Temple University School of Medicine, 1937.

William H. Swift, *Secretary.*

BRISTOL SOUTH DISTRICT

BARRETT, JAMES A., 7 Middle Street, South Dartmouth.
Maryland Medical College, 1910.

COSGROVE, THOMAS C., Main Street, Vineyard Haven.
College of Physicians and Surgeons, Boston, 1923.

DE MELLO, JOSEPH, 40 Bliss Street, South Dartmouth.
Tufts College Medical School, 1935.

DIAS, JOHN F., JR., 361 County Street, New Bedford.
Tufts College Medical School, 1935.

HARNEY, ALOYSIUS P., 167 Washington Street, New Bedford.
Harvard Medical School, 1934.

HORAN, GEORGE R., 560 Broadway, Fall River.
Georgetown University Medical School, 1935.

MUNCE, RICHARD T., Truesdale Hospital, Fall River.
Harvard Medical School, 1935.

PRIAL, DAVID, 104 East Main Street, Fall River.
St. Louis College of Physicians and Surgeons, 1926.

Albert H. Sterns, *Secretary.*

ESSEX NORTH DISTRICT

HUMPHREYS, STORER P., 155 High Road, Newbury.
Yale University School of Medicine, 1932.

MACLEOD, DOROTHY G., 60 High Street, Newburyport.
Tufts College Medical School, 1924.

SZOSTAK, RAYMOND G., 250 Prospect Street, Lawrence.
Kansas City University of Physicians and Surgeons, 1931.

Elmer S. Bagnall, *Secretary.*

ESSEX SOUTH DISTRICT

BABB, WARREN D., 40 Chestnut Street, Salem.
McGill University Faculty of Medicine, 1936.

CARROLL, WILLIAM J., Essex Sanatorium, Middleton.
Tufts College Medical School, 1935.

CUNNEY, JOHN V., 11½ Fowler Street, Salem.
Harvard Medical School, 1935.

GINSBERG, MAX, 314 Essex Street, Salem.
Tufts College Medical School, 1933.

JANNINO, EDMUND A., 843 Western Avenue, Lynn.
Middlesex College of Medicine and Surgery, 1933.

LARCHEZ, HENRY F., Walnut Road, South Hamilton.
Middlesex College of Medicine and Surgery, 1932.

OBERSON, HENRY J., 93 Eastern Avenue, Lynn.
Harvard Medical School, 1932.

REMILLARD, FLORA M., Danvers State Hospital, Hathorne.
University of Vermont College of Medicine, 1934.

RICHARDSON, RUSSELL R., 81 Highland Avenue, Salem.
Indiana University School of Medicine, 1933.

SPECTOR, NATHAN M., 8 Gray Circle, Lynn.
Tufts College Medical School, 1924.

TODD, BARNARD P., Beverly Hospital, Beverly.
Harvard Medical School, 1936.

WATTS, WINTHROP F., 2 Brookhouse Drive, Marblehead.
New York University College of Medicine, 1937.

J. Robert Shaughnessy, *Secretary.*

FRANKLIN DISTRICT

FOOTNICK, SAMUEL, 30 East Main Street, Orange.
Middlesex College of Medicine and Surgery, 1930.

KAYE, EDWARD, Mount Hermon.
Cornell University Medical College, 1936.

MOLOTCHICK, MAXWELL B., 29 Bridge Street, Millers Falls.
Middlesex College of Medicine and Surgery, 1928.

Charles Moline, *Secretary.*

HAMPDEN DISTRICT

BAILEY, EDWIN J., 499 Page Boulevard, Springfield.
Middlesex College of Medicine and Surgery, 1926.

COGAN, MICHAEL A., 13 Willow Street, Holyoke.
Vanderbilt University School of Medicine, 1936.

GATELEY, JOHN R., Mercy Hospital, Springfield.
Boston University School of Medicine, 1938.

GENEST, LEOPOLD O., 360 Main Street, Indian Orchard.
Boston University School of Medicine, 1937.

KEES, PHILIP A., 29 Mayfair Avenue, Springfield.
University of Minnesota Medical School, 1936.

KOCHANEK, JOSEPH M., 39 Ludlow Avenue, Indian Orchard.
Rush Medical College of University of Chicago, 1937.

LAZARCHICK, MICHAEL, 339 Front Street, Chicopee.
Middlesex College of Medicine and Surgery, 1933.

SHERMAN, DAVID E., 447 Sumner Avenue, Springfield.
Middlesex College of Medicine and Surgery, 1930.

STEINBERG, THEODORE, Providence Hospital, Holyoke.
Tufts College Medical School, 1938.

SYMINGTON, CLIFFORD L., 14 Chestnut Street, Westfield.
Philadelphia College of Osteopathy, 1928.
Middlesex College of Medicine and Surgery, 1932.

Hervey L. Smith, *Secretary*.

HAMPSHIRE DISTRICT

FAIRBANK, RUTH E., Mount Holyoke College, Woodbridge Terrace, South Hadley.
Johns Hopkins University School of Medicine, 1916.

PEKALA, JOSEPH G., 261 Main Street, Northampton.
University of Vermont College of Medicine, 1932.

Joseph D. Collins, *Secretary*.

MIDDLESEX EAST DISTRICT

BOYER, SAMUEL H., 77 Church Street, Winchester.
Tufts College Medical School, 1936.

CONDO, ANNUNZIATO, 1181-Company-V-CCC, North Reading.
Royal University of Naples, Italy, 1922.

LAND, HERBERT, North Reading.
Middlesex College of Medicine and Surgery, 1930.

MUELLER, HARRY L., 31 Church Street, Winchester.
Harvard Medical School, 1934.

POLAND, WARREN M., 230 Main Street, Wakefield.
Harvard Medical School, 1935.

Kenneth L. Maclachlan, *Secretary*.

MIDDLESEX NORTH DISTRICT

LAURIN, THEOPHILE, 169 Parkview Avenue, Lowell.
University of Bishop College Faculty of Medicine, Montreal, 1899.

Theodore A. Stamas, *Secretary*.

MIDDLESEX SOUTH DISTRICT

BAILEY, ORVILLE T., C-44 Dunster House, Cambridge.
Albany Medical College, 1932.

BARRON, ALBERT L., 666 Main Street, Watertown.
Middlesex College of Medicine and Surgery, 1928.

BUSKIRK, JAMES H., 38 Arlington Street, Cambridge.
Georgetown University Medical School, 1932.

CANZANELLO, VINCENT J., 385 Broadway, Everett.
Middlesex College of Medicine and Surgery, 1933.

COLANTINO, GEORGE J., 490 Highland Avenue, Malden.
Middlesex College of Medicine and Surgery, 1933.

DONALDSON, GORDON A., Lincoln.
Harvard Medical School, 1935.

GILLESPIE, LUKE, Malden Hospital, Malden.
Harvard Medical School, 1937.

GREGG, WARD I., Gerry's Landing, Cambridge.
Harvard Medical School, 1932.

HOLMES, JOSEPH A., 124 Walker Street, Cambridge.
Harvard Medical School, 1935.

HOPKINS, ANNE M., 1306 Massachusetts Avenue, Cambridge.
Columbia University College of Physicians and Surgeons, 1936.

INGALLS, THEODORE H., 48 Townsend Road, Belmont.
Harvard Medical School, 1933.

KAZANJIAN, KAREKAN A., 71 Langdon Avenue, Watertown.
Harvard Medical School, 1931.

KOZOL, HARRY L., 55 Rosalie Road, Newton Centre.
Harvard Medical School, 1934.

LAURENCE, MAURICE K., 84 Mandalay Road, Newton Centre.
Tufts College Medical School, 1937.

LYNCH, JOSEPH P., 330 Mt. Auburn Street, Cambridge.
Harvard Medical School, 1936.

MCCARTHY, CHARLES J., 466 Medford Street, Somerville.
College of Physicians and Surgeons, Boston, 1932.

MILLS, ABRAHAM A., 781 Moody Street, Waltham.
Middlesex College of Medicine and Surgery, 1928.

O'CONNOR, FRANK M., 277 Homer Street, Newton.
Boston University School of Medicine, 1937.

PROUT, CURTIS T., 163 Hillside Avenue, Arlington Heights.
Cornell University Medical College, 1924.

SOLOMON, LOUIS, 338 Ferry Street, Malden.
Middlesex College of Medicine and Surgery, 1933.

TIFFANY, GRACE E., 262 Irving Street, Framingham.
Tufts College Medical School, 1937.

UDIN, MIRIAM S., 29 Marlboro Street, Newton.
Middlesex College of Medicine and Surgery, 1925.

WATTLES, FRANK M., 24 Payson Road, Belmont.
Emory University School of Medicine, 1933.

WOOD, HAROLD, 1329 Walnut Street, Newton Highlands.
Tufts College Medical School, 1933.

ZETZEL, LOUIS, 20 Prescott Street, Cambridge.
Harvard Medical School, 1934.

Alexander A. Levi, *Secretary*.

NORFOLK DISTRICT

ALEXANDER, BENJAMIN, 210 Riverway, Roxbury.
Harvard Medical School, 1934.

ALLENDORF, FRANCIS J., 118 Common Street, Walpole.
Middlesex College of Medicine and Surgery, 1933.

ALPERT, GEORGE, 728 Morton Street, Dorchester.
University of Maryland School of Medicine and the College of Physicians and Surgeons, 1935.

BARTLETT, ESTHER E., New England Hospital for Women and Children, Roxbury.
University of Wisconsin Medical School, 1934.

Bell, Benjamin, 11 Crawford Street, Roxbury.
Jefferson Medical College of Philadelphia, 1936.

Bicchieri, Nunzio A., 474 Canterbury Street, Dorchester.
St. George's Hospital Medical School, 1935.

Bruce, Norman H., Pondville Hospital, Wrentham.
Harvard Medical School, 1934.

Chapman, Carrie E., 301 Chestnut Avenue, Jamaica Plain.
Tufts College Medical School, 1934.

Chase, Louis S., 453 Washington Street, Brookline.
University of Berlin, Germany, 1934.

Connor, John H. F., Emerald Street, Wrentham.
College of Physicians and Surgeons, Boston, 1907.

Cronan, Bernard P., 40 Pond Street, Sharon.
University of Kansas City, 1933.

Dillon, Raymond A., 97 Sewall Avenue, Brookline.
Harvard Medical School, 1938.

Di Rago, Joseph V., 44 York Terrace, Brookline.
Middlesex College of Medicine and Surgery, 1933.

Drooker, Joshua C., 15 Stratton Street, Dorchester.
Tufts College Medical School, 1933.

Duckering, Florence A., 4238 Washington Street, Roslindale.
Tufts College Medical School, 1937.

Dunphy, John E., 2 Netherlands Road, Brookline.
Harvard Medical School, 1933.

Feldman, Theodore, Robert Breck Brigham Hospital, Roxbury.
Tufts College Medical School, 1935.

Flynn, William F., 307 Edge Hill Road, Milton.
Yale University School of Medicine, 1933.

Gasson, Anthony W., 937 Washington Street, Norwood.
Middlesex College of Medicine and Surgery, 1934.

Glodt, Milton, 19 Thatcher Street, Brookline.
University of Basel, Switzerland, 1936.

Gorman, Lionel J., 364 Riverway, Roxbury.
Massachusetts College of Osteopathy, 1926.
Middlesex College of Medicine and Surgery, 1932.

Gross, Robert E., Peter Bent Brigham Hospital, Roxbury.
Harvard Medical School, 1931.

Hamlin, Edward, Jr., 1031 Brush Hill Road, Milton.
Harvard Medical School, 1933.

Harrison, James, 917 West Roxbury Parkway, Brookline.
Boston University School of Medicine, 1937.

Harrison, John H., 8 Brewster Terrace, Brookline.
University of Virginia Department of Medicine, 1932.

Hiscock, Mabelle C., New England Hospital for Women and Children, Roxbury.
Johns Hopkins University School of Medicine, 1935.

Hoffmann, Richard, 7 Egmont Street, Brookline.
University of Vienna, 1920.

Hogan, Joseph N., 39 South Street, Jamaica Plain.
College of Physicians and Surgeons, Boston, 1928.

Kevorkian, Albert Y., 47 Crehore Road, Chestnut Hill.
Tufts College Medical School, 1934.

Levin, Orrin, 1037 Beacon Street, Brookline.
Tufts College Medical School, 1935.

Levins, Leo V., 128 Longwood Avenue, Brookline.
Tufts College Medical School, 1936.

Luzackas, Pauline, New England Hospital for Women and Children, Roxbury.
Boston University School of Medicine, 1937.

Matarozzo, Mary K. D., 257 Belgrade Avenue, Roslindale.
Tufts College Medical School, 1924.

Moriarty, James E., 1074 South Street, Roslindale.
Middlesex College of Medicine and Surgery, 1933.

Norton, Harold F., 340 Canterbury Street, Dorchester.
Harvard Medical School, 1931.

Offenbach, Bertha, 9 Woodville Street, Roxbury.
Boston University School of Medicine, 1936.

Reidy, John A., 92 High Street, Brookline.
Harvard Medical School, 1934.

Scheidell, Dorothy K., New England Hospital for Women and Children, Roxbury.
Cornell University Medical College, 1932.

Solet, Leo, 280 Main Street, Franklin.
Middlesex College of Medicine and Surgery, 1933.

Sullivan, Daniel J., Boston State Hospital, Dorchester Center.
Temple University School of Medicine, 1934.

Sullivan, John F., Boston State Hospital, Dorchester Center.
Boston University School of Medicine, 1937.

Vickery, Eugene A., 6 Kenilworth Road, Wellesley.
Harvard Medical School, 1903.

Wagman, Ora H., 382 Riverway, Roxbury.
Tufts College Medical School, 1937.

Welch, Edward J., 20 Laurel Road, Milton.
Harvard Medical School, 1936.

Zalvan, Jacob, 64 North Street, Medfield.
Middlesex College of Medicine and Surgery, 1934.

Zetlin, Arnold, 72 Egmont Street, Brookline.
Tufts College Medical School, 1936.

Frank S. Cruickshank, *Secretary*.

NORFOLK SOUTH DISTRICT

Amrhein, Leo F., 774 Hancock Street, Quincy.
Tufts College Medical School, 1935.

Bozigian, Haig, Quincy City Hospital, Quincy.
Tufts College Medical School, 1938.

Carey, Edmund L., 59 South Street, Quincy.
Tufts College Medical School, 1935.

Dalton, Kenneth V., 133 Washington Street, Weymouth.
Tufts College Medical School, 1936.

D'Elia, Vincent, Quincy City Hospital, Quincy.
Tufts College Medical School, 1937.

Fox, Lester I., Quincy City Hospital, Quincy.
University of Maryland School of Medicine and College of Physicians and Surgeons, 1938.

Levin, Aaron H., Quincy City Hospital, Quincy.
Tufts College Medical School, 1937.

Lindblade, Eric H., 49 Whitney Road, Quincy.
Tufts College Medical School, 1919.

Millen, Morris H., 391 Bridge Street, North Weymouth.
Middlesex College of Medicine and Surgery, 1933.

Sherman, Julius, Quincy City Hospital, Quincy.
Tufts College Medical School, 1938.

Taylor, Morris, Quincy City Hospital, Quincy.
Boston University School of Medicine, 1938.

Walsh, Francis X., Quincy City Hospital, Quincy.
Tufts College Medical School, 1937.

Woodward, Appleton C., Norfolk County Hospital, South Braintree.
Tufts College Medical School, 1937.

Robert L. Cook, *Secretary*.

PLYMOUTH DISTRICT

Carr, William M., 463 Washington Street, Whitman.
Tufts College Medical School, 1935.

Giberti, Joseph V., 31 Oak Street, Middleboro.
Tufts College Medical School, 1937.

Pollen, David A., 24 School Street, Middleboro.
Middlesex College of Medicine and Surgery, 1932.

Ragonetti, Victor V., 145 Court St., Plymouth.
Middlesex College of Medicine and Surgery, 1931.

Howard C. Reed, *Secretary*.

SUFFOLK DISTRICT

Butts, Vincent, 244 Meridian Street, East Boston.
Kansas City University of Physicians and Surgeons, 1928.

Cassin, Benjamin I., 195 Chestnut Street, Chelsea.
Tufts College Medical School, 1931.

Edwards, Jesse E., 21 Worcester Square, Boston.
Tufts College Medical School, 1935.

Gersh, David H., Carney Hospital, South Boston.
Tufts College Medical School, 1937.

Ginsburg, Emanuel, 20 Garden Street, Boston.
Tufts College Medical School, 1933.

Guarciariello, Cosmo A., 131 Endicott Street, Boston.
Royal University of Rome, 1935.

Maloof, Frederic, 22 East Brookline Street, Boston.
Middlesex College of Medicine and Surgery, 1931.

Mansfield, James S., 33 Commonwealth Avenue, Boston.
Harvard Medical School, 1932.

Mavraides, William P., 194 Huntington Avenue, Boston.
University of Vermont College of Medicine, 1933.

McManus, John F., 818 Harrison Avenue, Boston.
Boston University School of Medicine, 1936.

Quigley, Thomas B., 24 Peterboro Street, Boston.
Harvard Medical School, 1933.

Roiff, Harry S., 159 Shurtleff Street, Chelsea.
St. Louis College of Physicians and Surgeons, 1923.

Ross, Michael M., 497 Broadway, South Boston.
Boston University School of Medicine, 1937.

Smith, Joseph T., 261 Beacon Street, Boston.
Johns Hopkins University School of Medicine, 1907.

Thibodeau, Arthur A., 77 Park Drive, Boston.
Tufts College Medical School, 1932.

Zollo, Felice J., 353 Revere Street, Revere.
Middlesex College of Medicine and Surgery, 1928.

John P. Monks, *Secretary*.

WORCESTER DISTRICT

Cicchetti, John R., 19 Church Street, Milford.
St. Louis University School of Medicine, 1934.

Corrado, Agostino L., 4 Park Street, Webster.
Long Island College of Medicine, 1933.

D'Argenis, Italia M., 22 Gage Street, Worcester.
Middlesex College of Medicine and Surgery, 1929.

Djerf, Charles, Worcester City Hospital, Worcester.
Tufts College Medical School, 1937.

Hostettler, Cleon W., 47 Summer Street, North Brookfield.
Middlesex College of Medicine and Surgery, 1933.

Lorge, Heinz, Rutland State Sanatorium, Rutland.
Johann Wolfgang Goethe-Universität Medical Faculty, 1933.

Nelson, Richard W., Grafton State Hospital, North Grafton.
McGill University Faculty of Medicine, 1935.

Render, Norman D., Worcester State Hospital, Worcester.
McGill University Faculty of Medicine, 1928.

Robinson, Roger W., 27 Elm Street, Worcester.
Northwestern University Medical School, 1935.

Russell, Frank H., 78 Burncoat Street, Worcester.
University of Tennessee College of Medicine, 1917.

Watson, Wilfred M., Memorial Hospital, Worcester.
Tufts College Medical School, 1937.

Weiksner, Francis A., 67 Providence Street, Worcester.
Middlesex College of Medicine and Surgery, 1929.

George C. Tully, *Secretary*.

WORCESTER NORTH DISTRICT

Grossman, Myer J., 599 Main Street, Athol.
Middlesex College of Medicine and Surgery, 1933.

Haimes, Solomon M., Gardner State Hospital, East Gardner.
Jefferson Medical College of Philadelphia, 1932.

Mattia, Anthony F., 97 Summer Street, Fitchburg.
College of Physicians and Surgeons, Boston, 1921.

Wasser, Louis, Elm Street, Baldwinsville.
Middlesex College of Medicine and Surgery, 1933.

Edward A. Adams, *Secretary*.

DEATHS

HART — Clarence D. Hart, M.D., of Somerville, died April 9 in Savannah, Georgia. He was in his forty-fifth year.

Dr. Hart received his degree from the Albany Medical College, New York, in 1931, and also attended Harvard Medical School. He had specialized in public health and had recently been appointed to serve as health officer of Savannah.

Dr. Hart was a member of the Massachusetts Medical Society and the American Medical Association.

His father survives him.

LYNCH — Charles E. Lynch, M.D., of Quincy, died April 13. He was in his forty-second year.

Born in South Boston he received his degree from Tufts College Medical School in 1923. He was a member of the Massachusetts Medical Society and the American Medical Association.

His widow, a son, a daughter, four sisters and a brother, Dr. James Lynch, survive him.

MISCELLANY

STAGE OF TUBERCULOSIS INFLUENCES PROGNOSIS

Hilleboe (Follow-up study of patients discharged from tuberculosis sanatoria. *Transactions of the Thirty-Fourth Annual Meeting of the National Tuberculosis Association, 1938*) succeeded in tracing 92.7 per cent of more than 5000 patients discharged from ten of the fifteen public tuberculosis sanatoriums in Minnesota during the ten-year period, 1926–1935. Patients studied were about equally divided between rural and urban residents. Of the total number about 36 per cent were dead on discharge. This tremendous loss gives some measure of the tragic toll taken by this disease even during hospitalization when expert medical attention and every facility for treatment are available. Living and dead are classified, according to stage of disease, as shown approximately in Chart 1.

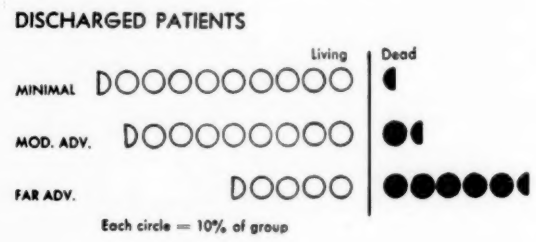

Chart 1.

Stage of disease influences the length of time needed for recovery. In this study all patients were in the sanatorium for ninety days or more. Living patients, not including those who were admitted more than once were

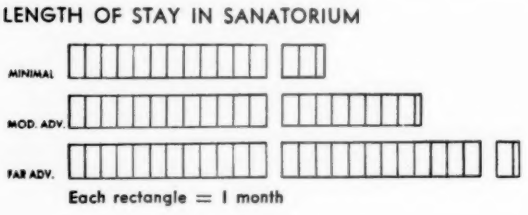

Chart 2.

classified according to the average length of stay in the sanatorium and the stage of the disease. Chart 2 pictures roughly the result.

The influence of stage of disease on the condition at the

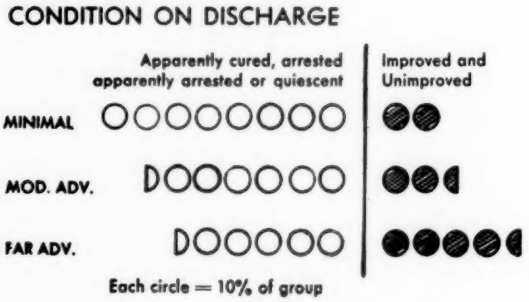

Chart 3.

time of discharge was studied and the results confirmed the observation that the early case has a much better chance of satisfactory recovery than has the advanced case. The result is summarized in Chart 3.

The probabilities of dying from any given disease can be calculated by actuaries with a fair degree of accuracy. In a person with tuberculosis the risk of dying is increased and this risk is in direct ratio to the stage of disease as shown in Chart 4.

"Statistical study of comparative mortality in discharged patients gives valid proof of the soundness of many clinical concepts regarding the disease. After all, one of the real values of statistics is to confirm the impressions of

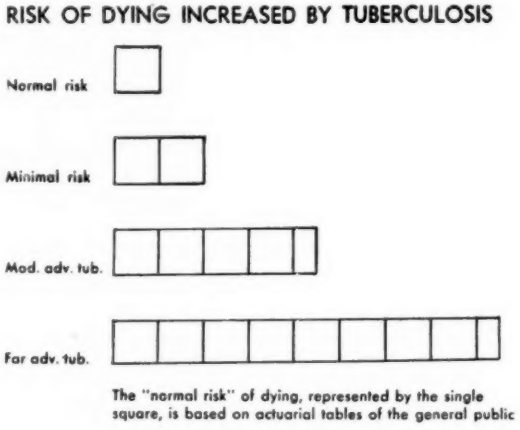
RISK OF DYING INCREASED BY TUBERCULOSIS
Normal risk
Minimal risk
Mod. adv. tub.
Far adv. tub.
The "normal risk" of dying, represented by the single square, is based on actuarial tables of the general public

Chart 4.

sound clinicians. Beneficial effects of early diagnosis of serious pulmonary tuberculosis lesions are reflected in the smaller risk of dying on the part of the minimal cases in comparison with the more advanced cases during the dangerous first five years after discharge. Tuberculosis must be diagnosed early." — Reprinted from *Tuberculosis Abstracts,* April, 1939.

RÉSUMÉ OF COMMUNICABLE DISEASES IN MASSACHUSETTS FOR FEBRUARY, 1939

DISEASES	FEB. 1939	FEB. 1938	FIVE-YEAR AVERAGE*
Anterior poliomyelitis	0	0	0
Chickenpox	1347	1607	1325
Diphtheria	12	10	25
Dog bite	580	510	492
Dysentery, bacillary	23	6	2
German measles	66	80	509
Gonorrhea	285	444	409
Lobar pneumonia	616	519	661
Measles	3825	811	3504
Meningococcus meningitis	7	8	11
Mumps	823	777	900
Paratyphoid B fever	2	1	0
Scarlet fever	899	1211	1000
Syphilis	343	462	425
Tuberculosis, pulmonary	164	141	221
Tuberculosis, other forms	15	23	28
Typhoid fever	5	5	5
Undulant fever	2	3	2
Whooping cough	1015	454	957

*Based on figures for preceding five years.

RARE DISEASES

Diphtheria was reported from: Athol, 1; Cambridge, 2; Haverhill, 1; Lawrence, 7; North Adams, 1; total, 12.

Dysentery, bacillary, was reported from: Danvers, 2; Lowell, 6; Wrentham, 15; total, 23.

Infectious encephalitis was reported from: Chicopee, 1; Westfield, 1; total, 2.

Meningococcus meningitis was reported from: Boston, 2; Leominster, 1; Lynn, 1; Newton, 1; Randolph, 1; Somerville, 1; total, 7.

Paratyphoid B fever was reported from: Brookline, 1; West Springfield, 1; total, 2.

Pellagra was reported from: Westminster, 1; total, 1.

Pfeiffer bacillus meningitis was reported from: Greenfield, 1; Lowell, 1; Springfield, 1; total, 3.

Septic sore throat was reported from: Boston, 5; Chelsea, 1; Fall River, 2; Framingham, 1; Greenfield, 1; Lawrence, 1; Lowell, 1; Lynn, 1; Malden, 2; New Bedford, 2; Quincy, 1; Somerville, 1; Woburn, 2; total, 21.

Trichinosis was reported from: Boston, 1; Springfield, 1; total, 2.

Typhoid fever was reported from: Boston, 1; Brookline, 1; Everett, 1; Lynn, 1; Plymouth, 1; total, 5.

Undulant fever was reported from: Andover, 1; Barnstable, 1; total, 2.

Whooping cough, measles, chickenpox, and paratyphoid B fever were reported above the five-year average.

Scarlet fever, diphtheria, mumps and German measles were reported below the five-year average.

Tuberculosis (other forms) showed record low figures for the second consecutive month.

Lobar pneumonia, meningococcus meningitis and pulmonary tuberculosis were reported below the five-year average.

Typhoid fever and undulant fever were reported at figures equal to that of the five-year average.

Animal rabies showed record low incidence for the third consecutive month. A new focus was noted in Woburn.

MAINE NEWS

The Women's Field Army

Plans for the 1939 campaign of the Women's Field Army of Maine are being completed under the direction of the Volunteer Campaign Committee consisting of Mr. Samuel Stewart, of Lewiston, chairman, Mrs. John H. Huddilston, of Orono, Mrs. William Holt, of Portland, Dr. Frederick T. Hill, of Waterville, and Dr. Edward H. Risley, of Waterville, chairman of the Advisory Board, ex officio.

April has been designated as Cancer-Control Month by the American Society for the Control of Cancer. The intensive campaign will be carried on in most communities in Maine during the week of April 3; although the entire month will be given over to the work for the benefit of those groups which cannot carry on their campaign during the first week. Continuing the fine co-operation of the medical profession with the Women's Field Army, ninety-eight physicians, representing all counties of the State, have accepted chairmanships of advisory boards in their various sections.

Figures compiled at headquarters show the scope of the service given in 1938 through the Dr. Joseph W. Scannell Memorial Fund. Every county is represented by patients treated from May to October, 1938, when, because of the increasing demand for free treatment, the funds became exhausted. As Maine has no hospital for the free treatment of the indigent cancer patient, this service of the Women's Field Army has been a great boon to the 194 needy patients treated by either radium or x-rays.

Postgraduate Education

The Committee on Graduate Education has prepared the following panel discussions which are now available for county-medical-society meetings:

Pneumonia — Dr. F. T. Hill, Waterville, chairman.

Cardiovascular Disease — Dr. E. E. Holt, Jr., Portland, chairman.

Laboratory Procedures and Their Relation to Clinical Medicine — Dr. Julius Gottlieb, Lewiston, chairman.

The Acute Surgical Abdomen — Dr. F. H. Jackson, Houlton, chairman.

Fractures — Dr. Allan Woodcock, Bangor, chairman.

Antipneumococcus Serum

The following facts relative to the availability of therapeutic serum in the different types of pneumonia have been recently announced by the State Board of Health and Welfare.

Therapeutic antipneumococcus serum is now available for Types 1, 2, 4, 5, 6, 7, 8 and 14. Typing stations are supplied with Types 1, 2, 5 and 7. Types 4, 6, 8 and 14 are available at the Augusta office, and will be supplied when a messenger is sent for them or will be shipped by first-class mail or express. It is eventually intended to supply Portland, Lewiston, Waterville, Bangor and Caribou with all available types. Types 3, 9, 11, 13, 18, 19, 20, 23, 28 and 29 are available only on special order from New York for individual cases, so no messenger should be sent to Augusta for these types. One may wire Augusta for the above special types. Other types are not available at present.

Serum is supplied free to indigent patients; those who can pay will be billed at cost. Relatives or neighbors should transport the specimens. Regular messenger service is not available in Augusta, and such transportation is not a regular function of the State Police.

Notes

The following members of the Maine Hospital Association appeared before the Budget Committee of the Legislature on November 29, 1938, and presented reasons why it was necessary for hospitals to receive more money for the care of the indigent sick: Mr. Samuel Stewart (chairman), president, Central Maine General Hospital, Lewiston; Mr. Robert Braun, president, Maine General Hospital, Portland; Mr. George Eaton, president, Eastern Maine General Hospital, Bangor; Mr. Carroll Perkins, Thayer Hospital, Waterville; Dr. Stephen Brown, superintendent, Maine General Hospital, Portland; Dr. Allan Craig, medical director, Eastern Maine General Hospital, Bangor; Dr. Joelle C. Hiebert, president, Maine Hospital Association, Lewiston. The committee was cordially received by the Budget Committee, and it is hoped that larger appropriations will be granted shortly. This committee also prepared a lien bill for hospital service only.

The osteopaths are sponsoring two bills which are designed to compel general hospitals to give them the right to practice in them. The Maine Hospital Association plans to fight these bills vigorously.

Dr. Samuel Levine, of Boston, was the guest speaker at the April 20 meeting of the Kennebec County Medical Society at the Gardiner General Hospital, Gardiner.

CORRESPONDENCE

ADMISSIONS TO STATE BOARD EXAMINATIONS

To the Editor: I am enclosing a copy of the preliminary report of the Board of Registration in Medicine on admissions to the March, 1939, examination.

Stephen Rushmore, M.D., *Secretary.*
Board of Registration in Medicine,
State House, Boston.

Preliminary Report of the Board of Registration in Medicine on Admissions to March, 1939, Examinations

One hundred and ninety-three candidates were examined. They can be arranged in three groups according to the school from which they graduated as follows: approved schools, non-approved schools, osteopathic schools. Each group may be further subdivided into those taking the examination for the first time; those taking it for the second or third time; those taking it for the fourth or more time. Candidates who fail three times within one year must make out a new application which places them in the last group. Table 1 gives the figures in summary form.

Table 1.

	Approved		Non-Approved	Osteopathic	Total
	North American	Foreign			
First examination	36	16	16	5	73
Second or third examination	0	8	65	12	85
Fourth or more examination	0	2	31	2	35
Totals	36	26	112	19	193

In Table 2 the number of times the applicants had taken their examinations is arranged according to the school from which they graduated. The order of the schools is chronological according to the date on which the first graduate of each school filed application for this examination.

Table 2.

School	First Examination	Second or Third Examination	Fourth or More Examination	Total
Middlesex College of Medicine and Surgery	10	39	19	68
Kansas City University of Physicians and Surgeons	0	12	7	19
University of Rome	0	1	0	1
College of Physicians and Surgeons (Boston)	3	6	3	12
University of Naples	0	1	0	1
Mid-West Medical College	1	7	2	10
Massachusetts College of Osteopathy	1	5	2	8
Syracuse University	1	0	0	1
Kirksville College of Osteopathy	3	5	0	8
Philadelphia College of Osteopathy	1	2	0	3
University of Vienna	8	1	0	9
Missouri College of Medicine and Science	0	1	0	1
University of Ghent	0	1	0	1
University of Munich	1	0	0	1
University of Tartu	0	1	0	1
University of Bonn	0	1	0	1
University of Berlin	1	1	0	2
Royal Colleges (Edinburgh)	0	1	0	1
Chicago Medical School	1	0	0	1
Boston University School of Medicine	3	0	0	3
University of Heidelberg	2	0	0	2
McGill University	2	0	0	2
Harvard Medical School	4	0	0	4
Columbia University	2	0	0	2
Tufts College Medical School	7	0	0	7
Hahnemann Medical School	1	0	0	1
New York University	1	0	0	1
University of Nebraska Medical School	1	0	0	1
Creighton University Medical School	1	0	0	1
University of Michigan	2	0	0	2
University of Lausanne	1	0	1	2
Washington University	1	0	0	1
University of Wurzburg	0	0	1	1
Fordham University Medical School	1	0	0	1
Marquette Medical School	1	0	0	1
University of Western Ontario	1	0	0	1
University of Prague	1	0	0	1
Rochester University	1	0	0	1
Des Moines Still College of Osteopathy	1	0	0	1
Emory Medical School	1	0	0	1
Northwestern University	2	0	0	2
Long Island Medical School	1	0	0	1
Bowdoin Medical School	1	0	0	1
University of Geneva	1	0	0	1
Jefferson University	1	0	0	1
University of Leipzig	1	0	0	1
Totals	73	85	35	193

Comment

There were in this examination no repeaters among the graduates of approved schools in the United States and Canada.

Of a total of 193 candidates examined, 120 were repeaters.

Of a total of 193 candidates examined, 112 were from non-approved schools, reported as not eligible for admission to examination in any other state, and 19 osteopathic candidates for whom there are variations in the requirements of other states.

Of 52 graduates of approved schools taking the examination for the first time, 16 or about one third were from European universities (chiefly Continental).

UNAUTHORIZED SPONSORSHIP

To the Editor: I am informed that funds are being solicited for a "Grand Ball to Fight Communism," sponsored by a list to which my name and the names of other employees of the Boston Health Department are attached.

I know nothing of this affair and the use of my name is unauthorized.

Frederick J. Bailey, M.D.,
Deputy Health Commissioner.

City Hall Annex,
Boston.

ARTICLES ACCEPTED BY THE AMERICAN MEDICAL ASSOCIATION COUNCIL ON PHARMACY AND CHEMISTRY

To the Editor: In addition to the articles enumerated in our letter of March 9 the following have been accepted:

Campbell Products, Inc.

Ampules Mercupurin, 1 cc.
Ampules Mercupurin, 2 cc.

Eli Lilly & Co.

Ampules Solution Liver Extract Purified, 1 cc. — Lilly, 15 U.S.P. units per cc.

Sharp & Dohme

Sulfanilamide Tablets, 7½ gr.

E. R. Squibb & Sons

Concentrated Antipneumococcic Serum — Squibb, Types 5 and 7
Concentrated Antipneumococcic Serum — Squibb, Types 4 and 8

The Upjohn Co.

Tablets Cinchophen, 5 gr.
Tablets Cinchophen, 7½ gr.

Winthrop Chemical Co., Inc.
Tablets Suprarenin, 0.02 gm.

PAUL NICHOLAS LEECH, *Secretary.*

535 North Dearborn Street,
Chicago, Illinois.

REPORT OF MEETING

WILLIAM HARVEY SOCIETY

At a meeting of the William Harvey Society of Tufts College Medical School on Friday, February 10, in the Beth Israel Hospital auditorium, Dr. H. E. MacMahon introduced the speaker of the evening, Dr. William Boyd, professor of pathology and bacteriology at the University of Toronto. Dr. Boyd discussed the subject "Nephritis: The basis of the clinical picture" in a style and manner familiar to all recent medical students and graduates who have enjoyed his textbooks.

Dr. Boyd began by describing the normal kidney and its function. The glomeruli (renal filters) and the tubules (concentrating mechanisms) go to make up the nephron, which is the unit of structure. In the glomeruli, proteins and colloids are held back in the blood stream, while crystalloids are allowed through; in the tubules, the absorption of water, sugar and chlorides takes place, and the glomerular filtrate is thus concentrated to form urine. When absorption fails, an unconcentrated urine results; but if the tubular epithelium fails completely and disappears, then by experimental work in the tracing of the course of certain dyes it has been shown that the glomerular filtrate is wholly absorbed back into the tissues surrounding the tubules and anuria results. Anuria is, then, not a suppression of urine but a complete reabsorption of it. This has been shown to occur in cases with corrosive sublimate poisoning.

The glomerular filter can be described as a hand, the efferent and afferent arterioles forming the wrist and palm, the individual capillary loops, the fingers. The essential structure is the capillary "finger" projecting into the glomerular space. There are three elements to this capillary: a basement membrane; an endothelium consisting of cells spaced at intervals; and an epithelium consisting of a continuous layer of cells. A differential stain is necessary in demonstrating these elements. The key to Bright's disease is this capillary loop.

Dr. Boyd illustrated with lantern slides some remarkably clear-cut tissue sections of the kidney. The efferent arteriole from the glomerulus goes to the tubule, but its complete destruction will not result in tubular ischemia since a shunt exists, called "Ludwig's vessel."

From this, one might conclude that the glomerulus is the sine qua non of kidney function, but there are certain fishes whose kidneys have no glomeruli. Similarly, in advanced Bright's disease one can demonstrate nephrons consisting of healthy tubules and no glomeruli. These tubules must necessarily do the secreting for the absent glomeruli, but since they cannot secrete and absorb at the same time, the latter function is given up, and the result is an unconcentrated urine. Proof positive of Bright's disease is a low, fixed specific gravity for hours or days. Casts, cells, albumin, and so forth, are not pathognomonic but merely incidental. Pathologically, in advanced Bright's disease the most striking finding in sections examined microscopically is atrophy of the tubules.

Bright's disease is best classified as of two types. One is a true glomerulonephritis; the other, given various names, results in the same picture but arrives at it through a narrowing of the arterioles. Glomerulonephritis is a non-suppurative inflammation; there are no foci of suppuration, and the lesion is so disseminated as to suggest the action of a diffusible toxin, nearly always of streptococcal origin, from the nose, throat or nasopharynx. However, two great groups of poisons can cause the lesion: crystalloids — chemicals — pass the glomerular filter so easily as not to harm it, but on concentration in the tubules the poison becomes strong enough to damage the tubular epithelium; colloids, such as bacterial toxins, are of much larger molecular size and are therefore concentrated in the glomerular filter and produce damage there. As a result of this, the power of the glomerulus to hold back colloids is lost and thus the toxins are allowed to pass on and produce secondary damage to the tubules.

In glomerulonephritis there is exudation but not suppuration. The glomerular tuft becomes far more cellular in appearance than normal, and this turns out, by differential staining, to be an endothelial proliferation. It is the first indication of the onset of ischemia; the loop narrows and eventually becomes occluded by hyaline thrombus formation. With the cutting off of blood supply, the filter is damaged and exudation of blood occurs into the glomerular space.

In hospital practice the sequence of events is not clearly seen because the patient's lifetime disease is often treated at various institutions. There are three stages: acute, subacute and chronic. In the acute stage the diffusible toxin in the circulation acts on all the glomeruli. As a rule the patient rarely dies at this stage but goes on to the next stage. Obviously, some of the glomeruli must recover, if the patient does, and the kidney sinks down and down only as recurring attacks take toll of the remaining ones. The patient who dies in the subacute stage has a large, white kidney, which is classically associated with the edematous, anemic patient. The kidney cortex is swollen, and there are doubly refractile lipoid bodies in the epithelial cells. If the patient recovers from this stage, however, he goes on to the chronic stage, in which, post mortem, one sees loss of glomeruli, aglomerular tubules and a contracted kidney. Some healthy nephrons remain, which have carried the patient along, and these account for the gross picture of a nodular, scarred kidney. Microscopically, the striking thing is a disappearance of the normal number of renal tubules.

The other main type of Bright's disease is variously termed the "arteriosclerotic," "nephrosclerotic" or "hypertensive" kidney, the essential lesion being thickening of the afferent arteriole and narrowing of its lumen so as to produce ischemia. Thus the same end result is obtained but by a different approach. This is the kidney of hypertension. Which is first, the hypertension or the arteriolar narrowing, is an academic question; Dr. Boyd believes that hypertension is primary.

Dr. Boyd made further comment on some of the principal symptoms of nephritis. (1) Albuminuria is the result of damage to the renal filter causing increase in its permeability. Tissue sections show this by the presence of coagulum in the capsular space. (2) Edema in the subacute stages is due to the failure of the osmotic-pressure relations maintained by plasma colloids, which are lost in the urine. It is logical, therefore, that the first symptom be albuminuria, the second, edema. (3) Casts in the urine signify the large amount of albuminous material concentrated in the tubules so as to form molds. They incorporate whatever debris there is. (4) Inflammatory cells are present, of course, because this is an inflammation. Damaged epithelial cells are cast off. (5) With great damage and blocking, waste substances (nonprotein nitrogen)

are inefficiently eliminated. Bright himself suspected this, and confirmed the suspicion by having a chemist determine the blood urea. (6) The rise in blood pressure, together with its mechanism, is a story in itself.

Dr. Boyd presented a chart showing the relation of symptoms to lesions. The glomerulus, through damage to the basement membrane, is responsible for albuminuria, low plasma proteins and edema; through its capillary endothelium, it gives rise to hematuria, hypertension and renal insufficiency. Damages to the tubules results in a loss of concentrating power and, hence, a urine of low specific gravity.

In conclusion, Dr. Boyd suggested that a few beacons do stand out in the sea of perplexing problems concerning glomerulonephritis; these are: damage to the renal filter, damage to the absorbing mechanism and ischemia.

NOTICES

REMOVAL

Moses J. Stone, M.D., announces the removal of his office to 520 Beacon Street, Boston.

PHYSICIAN-IN-CHIEF, PRO TEMPORE, OLD HOME WEEK

During the week commencing Monday, April 24, a Physician-in-Chief, Pro Tempore, Old Home Week will be held at the Peter Bent Brigham Hospital. Those participating, each of whom has at some time served as physician-in-chief, pro tempore, at the hospital, include: Dr. Lewellys F. Barker, Baltimore, professor of medicine, emeritus, Johns Hopkins University; Dr. Francis G. Blake, New Haven, Sterling Professor of Medicine, Yale University; Dr. Alvah H. Gordon, Montreal, associate professor of medicine, McGill University; Dr. Duncan Graham, Toronto, professor of medicine, University of Toronto; Dr. James B. Herrick, Chicago, professor of medicine, emeritus, Rush Medical College; Dr. Warfield T. Longcope, Baltimore, professor of medicine, Johns Hopkins University; Dr. O. H. Perry Pepper, Philadelphia, professor of medicine, University of Pennsylvania; Dr. David Riesman, Philadelphia, professor of clinical medicine, emeritus, University of Pennsylvania, and Dr. Rollin T. Woodyatt, Chicago, clinical professor of medicine, University of Chicago. The program is as follows:

WARD ROUNDS: 10–12

Monday, April 24. Drs. Pepper (leader), Barker, Gordon, Riesman, Woodyatt.

Tuesday, April 25. Drs. Riesman (leader), Barker, Blake, Gordon, Graham, Longcope, Pepper and Woodyatt.

Wednesday, April 26. Drs. Longcope (leader), Barker, Blake, Gordon, Graham, Riesman and Woodyatt.

Thursday, April 27. Drs. Herrick (leader), Gordon, Blake, Graham, Riesman and Woodyatt.

Friday, April 28. Drs. Woodyatt (leader), Gordon and Riesman.

Saturday, April 29. Staff Rounds. Drs. Gordon (leader), Riesman, Woodyatt and Christian and the medical staff of the Peter Bent Brigham Hospital.

AMPHITHEATER CLINICS: 3:15–4:45

Monday, April 24. Drs. Barker and Riesman.

Tuesday, April 25. Drs. Longcope and Pepper.

Wednesday, April 26. Drs. Herrick and Gordon.

Thursday, April 27. Drs. Graham and Woodyatt.

Friday, April 28. Dr. Christian.

The afternoon amphitheater clinics are open to all physicians and students who may care to attend.

HOSPITAL RESEARCH COUNCIL

The next meeting of the Hospital Research Council will be held in the Ether Dome of the Massachusetts General Hospital, on Tuesday, April 25, at 5:00 p. m.

PROGRAM

Vitamin C Lack After Major Surgery. Dr. C. M. Jones.

Prothrombin Determination. Dr. J. D. Stewart.

Student Health Problems in Colleges. Dr. A. V. Bock.

Activity in the Cerebral Cortex During Anesthesia. Dr. H. K. Beecher.

Henry K. Beecher, M.D., *Secretary.*

MEDICAL CLINIC AT THE PETER BENT BRIGHAM HOSPITAL

At 3:30 p. m. on Thursday, April 27, in the amphitheater of the Peter Bent Brigham Hospital, Dr. James B. Herrick will give a medical clinic. Practitioners and medical students are cordially invited to attend.

SIR WILLIAM OSLER HONOR SOCIETY

Dr. Abraham Myerson, professor of clinical psychiatry at Harvard Medical School, will deliver a lecture, illustrated by slides, on "Human Autonomic Pharmacology" at a meeting of the Sir William Osler Honor Society of the Middlesex University School of Medicine, to be held in the auditorium at 415 Newbury Street, Boston, at 8:00 p. m. on April 26. The medical profession is cordially invited to attend.

JOINT MEETING OF THE SUFFOLK DISTRICT MEDICAL SOCIETY AND THE BOSTON MEDICAL LIBRARY

A joint meeting of the Suffolk District Medical Society and the Boston Medical Library will be held on Wednesday evening, April 26, at 8:15 p. m., at the Boston Medical Library, 8 Fenway, Boston.

PROGRAM

Official reports of the Society for 1938.

Election of officers.

Science and the Art of Deception. Dr. Francis G. Benedict.

Ladies are cordially invited to attend this meeting.

James M. Faulkner, M.D., *Secretary,*
Boston Medical Library.

John P. Monks, M.D., *Secretary,*
Suffolk District Medical Society.

SUFFOLK CENSORS' MEETING

The censors of the Suffolk District Medical Society will meet for the examination of candidates at the Boston Medical Library, 8 Fenway, Boston, on Thursday, May 4, at 4:00 p. m.

Candidates should make personal application to the secretary and present their medical diplomas at least one week before the examination.

John P. Monks, M.D., *Secretary.*

MIDDLESEX SOUTH DISTRICT MEDICAL SOCIETY

The annual meeting of the Middlesex South District Medical Society will be held at the Hotel Continental,

Cambridge, on Wednesday, May 3. The business meeting will begin at 11:30 a. m. At noon the annual oration will be delivered by Dr. Roy D. Halloran on "Opportunities of the Psychiatric Hospital in the Mental Health Problem."

Members in good standing are invited to the luncheon at 12:45 p. m.

FRED R. JOUETT, M.D., *President,*
ALEXANDER A. LEVI, M.D., *Secretary.*

NEW ENGLAND SOCIETY OF PSYCHIATRY

The annual meeting of the New England Society of Psychiatry will be held at Kline Memorial Hall, Metropolitan State Hospital, Waltham, on Tuesday, April 25, at 12 o'clock noon. Inspection of the hospital will be followed by a luncheon and business meeting. Dr. John W. Thompson and Dr. William Corwin will speak on "Some Observations on Patients Diagnosed as Having Schizophrenia."

NEW ENGLAND SOCIETY OF PHYSICAL MEDICINE

The regular meeting of the New England Society of Physical Medicine will be held at the Hotel Kenmore, Boston, on Wednesday evening, April 26, at 7:30 p. m. Dinner will be served at 6:00 p. m.

PROGRAM

Back Strain. Dr. Claude L. Payzant.

Treatment of Flat Feet. Dr. Howard Moore.

Nerve Trauma. Dr. Gordon M. Morrison.

Therapeutic Exercises in Bursitis of the Shoulder. Lucy G. Marshall.

General discussion.

All members of the medical profession are cordially invited to attend.

WILLIAM D. McFEE, M.D., *Secretary.*

SOCIETY MEETINGS AND CONFERENCES

CALENDAR OF BOSTON DISTRICT FOR THE WEEK BEGINNING MONDAY, APRIL 24

TUESDAY, APRIL 25

*9-10 a. m. Here and There in Endocrinology. Dr. Fuller Albright. Joseph H. Pratt Diagnostic Hospital.

*10 a. m. - 12:30 p. m. Tumor clinic. Boston Dispensary.

5 p. m. Hospital Research Council. Ether Dome, Massachusetts General Hospital.

*8:15 p. m. Harvard Medical Society. Amphitheater, Peter Bent Brigham Hospital (Shattuck Street entrance).

WEDNESDAY, APRIL 26

*9-10 a. m. Hospital case presentation. Dr. S. J. Thannhauser. Joseph H. Pratt Diagnostic Hospital.

*12 m. Clinicopathological conference. Children's Hospital amphitheater.

*7:30 p. m. New England Society of Physical Medicine. Hotel Kenmore, Boston.

*8 p. m. Sir William Osler Honor Society. Middlesex University School of Medicine auditorium, 415 Newbury Street, Boston.

*8:15 p. m. Joint meeting of the Suffolk District Medical Society and the Boston Medical Library. Boston Medical Library.

THURSDAY, APRIL 27

8:30-9:30 a. m. Exchange visit, Surgical and Orthopedic Staffs of the Peter Bent Brigham and Children's hospitals, held this week at the Children's Hospital Orthopedic.

*9-10 a. m. Alcohol: Chemical tests for alcoholism. Dr. Sydney Selesnick. Joseph H. Pratt Diagnostic Hospital.

*3:30 p. m. Medical clinic at the Peter Bent Brigham Hospital.

FRIDAY, APRIL 28

*9-10 a. m. Heredity and Environment in Relation to Intelligence, Personality and Mental Disease. Dr. Abraham Myerson. Joseph H. Pratt Diagnostic Hospital.

*10 a. m. - 12:30 p. m. Tumor clinic. Boston Dispensary.

12 m. Clinical meeting of the Children's Medical Service. Massachusetts General Hospital. Ether Dome.

*4 p. m. New England Heart Association, House of the Good Samaritan. 6:30 p. m., annual dinner, Harvard Club. 8:15 p. m., Boston Medical Library.

SATURDAY, APRIL 29

*9-10 a. m. Hospital case presentation. Dr. S. J. Thannhauser. Joseph H. Pratt Diagnostic Hospital.

*10 a. m. - 12 m. Staff rounds of the Peter Bent Brigham Hospital.

*Open to the medical profession.

APRIL 21 — New England Roentgen Ray Society. Page 649, issue of April 13.

APRIL 21 and 22 — New England Health Education Institute. Page 614, issue of April 6.

APRIL 23 — Health Lecture, Quincy City Hospital. Page 636, issue of February 23.

APRIL 25 — Harvard Medical Society. Page 649, issue of April 13.

APRIL 25 — New England Society of Psychiatry. Notice above.

APRIL 25 — Hospital Research Council. Page 688.

APRIL 26 — Sir William Osler Honor Society. Page 688.

APRIL 26 — New England Society of Physical Medicine. Notice above.

APRIL 27 — Medical clinic. Peter Bent Brigham Hospital. Page 688.

APRIL 28 — New England Heart Association. Page 649, issue of April 13.

MAY 3-6 — American Association of Mental Defect. Page 614, issue of April 6.

MAY 7-15 — International Congress of Military Medicine and Pharmacy. Page 501, issue of September 29.

MAY 11 — Pentucket Association of Physicians, 8:30 p. m., Hotel Bartlett, 95 Main Street, Haverhill.

MAY 12 and 13 — American Heart Association. Page 542, issue of March 23.

MAY 13-16 — American Board of Obstetrics and Gynecology. Page 457, issue of March 9.

MAY 14-20 — American Physicians' Art Association. Page 404, issue of March 2.

MAY 15-19 — American Medical Association. St. Louis, Missouri.

MAY 22, 23, and 24 — American Association for the Study of Goiter. Page 405, issue of March 2.

JUNE 5, 6, 7, and 8 — American Association of Industrial Physicians and Surgeons. Page 581, issue of March 30.

JUNE 6, 7, and 8 — Massachusetts Medical Society. Worcester.

JUNE 12-17 — Symposium on the Public-Health Significance of the Virus and Rickettsial Diseases. Page 125, issue of January 19.

JUNE 26-29 — National Tuberculosis Association. Page 936, issue of December 8.

SEPTEMBER — Boston Psychoanalytic Institute. Page 450, issue of September 22.

SEPTEMBER 11-15 — American Congress on Obstetrics and Gynecology. Page 938, issue of December 8.

SEPTEMBER 15-28 — Pan-Pacific Surgical Association. Page 863, issue of November 24.

OCTOBER 23 - NOVEMBER 3 — New York Academy of Medicine. Page 581, issue of March 30.

FALL, 1939 — Temperature Symposium. Page 218, issue of February 2.

DISTRICT MEDICAL SOCIETIES

ESSEX SOUTH

MAY 10 — Page 649, issue of April 13.

MIDDLESEX SOUTH

MAY 3 — Page 688.

SUFFOLK

APRIL 26 — Page 688.

MAY 4 — Censors' meeting. Page 688.

WORCESTER

MAY 10 — Worcester Country Club — annual meeting.

BOOK REVIEWS

Shock and Related Capillary Phenomena. Virgil H. Moon. 442 pp. London, New York and Toronto: Oxford University Press, 1938. $3.50.

This stimulating monograph represents the contribution of a pathologist to the solution of the problem of the etiology and mechanism of shock. The rather strange but apparently undeniable fact is pointed out that the vast

literature on this problem has emanated almost solely from surgeons and physiologists, without the corroborative evidence available in morphologic pathology, which, it is believed, furnishes convincing proof that shock is due to diminished blood volume and flow dependent on the leakage of plasma into the tissues as a result of increased permeability of the capillary endothelium and on an enormous increase in the capacity of the capillary bed, especially in visceral areas, with consequent stagnation and increased concentration and viscosity of the blood. The chief theories variously ascribing the universally admitted circulatory failure of shock to a weakened myocardium, to exhaustion of a vasomotor nerve center, to a failing venopressor mechanism, to decreased alkali reserve and acapnia and to hypothetical poisonous substances liberated from injured tissues are critically reviewed and evaluated, and their apparently untenable features pointed out. The structure and function of capillaries, — normal and pathologic, — as revealed in recent studies, are described, and the fact emphasized that they are endothelial tubes capable of contraction and dilatation, possibly under vasomotor control but certainly under the influence of substances freed from tissue cells suffering from anoxia. This is the key to the vicious circle of shock: tissue anoxia, dilatation and increased permeability of capillaries, stagnation and concentration of blood, increased tissue anoxia. It is pointed out that the circulatory failure characterizing a wide variety of conditions — burns; anaphylactic shock; poisoning by food, bee-sting, snake venom, peptones or various mineral agents; metabolic intoxications; acute infections; acute abdominal emergencies and peritonitis — is due to the same alteration of capillary function. Congestion of the viscera is regularly recorded in the necropsy records of all these conditions and is usually interpreted as "passive congestion due to heart failure," whereas the condition is actually acute capillovenous congestion and congestive circulatory failure, which are not to be ascribed to myocardial weakness except in cases of demonstrable cardiac defect or degeneration.

Dr. Moon's monograph is not lacking in practical applications of his theory of shock. He points out that hemoconcentration, which is fairly easily demonstrable by simple methods, occurs before any marked fall of blood pressure, which is maintained by the efficiency of the myocardium and the vasoconstrictor center; this is the earliest detectable manifestation of shock and the most accurate index of its severity. He discusses the logic of various therapeutic methods and emphasizes the primary need to restore blood volume and capillary tonus and to correct anoxia. He has succeeded admirably in integrating the mass of factual information about shock — derived from more than 400 titles in the appended bibliography. Doubtless it will be a long time before the last word is written about shock, but Dr. Moon's discriminating review of the literature and his application of the evidence afforded by pathology to the solution of the problem constitute an important contribution.

Diagnostic Standards: Tuberculosis of the lungs and related lymph nodes. 32 pp. Tentative edition. New York: National Tuberculosis Association, 1938.

This pamphlet, which is the eleventh in a series published by the National Tuberculosis Association, is a tentative one, and hence comments are requested. So long as the diagnosis and treatment of pulmonary tuberculosis undergo changes, so long will our conceptions and standards have to be revised to conform with the best opinions of the day.

It is noteworthy that the terms "childhood type" and "adult type" have been replaced by "primary tuberculosis" and "reinfection tuberculosis" respectively. Although the latter terms hardly describe the true underlying lesions, as no infection is strictly primary and one cannot draw the line between primary infection and reinfection, nevertheless they give a better concept of the pathologic changes than did the earlier terms.

Another section worth mentioning is the paragraph defining the minimum standard for a "negative sputum." This is no longer a haphazard term, but requires that at least three adequate specimens per month shall have been found to be negative, both by direct smear and by concentration. For patients to be designated as "apparently cured," the sputum must remain negative to repeated cultures and animal inoculations.

The reviewer wonders why nothing was said about the erythrocytic sedimentation rate and differential leukocytic studies. It appears to be universally accepted that a case cannot be designated as apparently cured unless the sedimentation rate is within normal limits. It is hoped that the above laboratory studies will be included in the next edition.

Control of Conception. Robert L. Dickinson. Second edition. 390 pp. Baltimore: Williams & Wilkins Co., 1938. $3.50.

In his book the author has gone into the whole subject of the control of conception in a most systematic, painstaking manner. The effectiveness of the various types of prevention is thoroughly discussed, and explained in great detail. The entire book is carefully illustrated by his own drawings. For those physicians who want a complete, authoritative work on the subject of contraception, this book will prove very satisfactory.

The laity has so many times confused the control of conception with abortion that it seems to the reviewer inadvisable for Dr. Dickinson to have included the indications and technic of abortion in this otherwise excellent book.

A Manual of Reparative Plastic Surgery. J. Eastman Sheehan. 311 pp. New York and London: Paul B. Hoeber, Inc., 1938. $5.50.

Since the World War there has been a growing interest in reparative plastic surgery. As a result, in addition to papers appearing in surgical journals, many books have been published on the subject. This interest is undoubtedly due to an increase of traumatic injuries from automobile and other civilian accidents.

Dr. Sheehan's book is printed in good type. It is profusely illustrated with diagrams and photographs which are very helpful. The author has divided the book into two parts. The first is directed to general consideration of principles of plastic repair, while the second describes various plastic operations.

A more critical survey indicates that, while certain subjects, such as plastic operations on the eyelids and the nose, are well covered, other sections are quite incomplete. This is particularly true concerning mandibular defects and reconstructive problems.

The point is rapidly being reached at which no single book on plastic surgery will be anything more than an introduction to the subject. The field as a whole has become unusually wide, and a book of the sort being reviewed, aside from providing a panorama of high lights, has its chief value in presenting the author's own particular clinical contributions and methods of procedure. In this last respect this volume should prove of value.